THE PLANT DISEASE REPORTER

Issued By

CROPS RESEARCH DIVISION

AGRICULTURAL RESEARCH SERVICE

UNITED STATES DEPARTMENT OF AGRICULTURE

LIBRARY
RECORD
★ DEC 16 1959 ★
U. S. DEPARTMENT OF AGRICULTURE

Volume 43 **Number** 12

December 15, 1959

The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Crops Research Division serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

SUGGESTIONS FOR PREPARATION OF MANUSCRIPTS FOR THE PLANT DISEASE REPORTER

(1) GENERAL: The Reporter page measures 9 inches long with the heading or 8 3/4 inches for the text part, by 6 inches wide. The copy is typed on a larger page, 11 1/4 inches of text or 12 inches overall in length by 8 inches in width, and reduced 25 percent in the photographic process of reproduction. Illustrations or tables larger in either dimension will take a correspondingly greater reduction. Only one size of type is available for text, footnotes, or tables.

(2) MANUSCRIPTS should be the original ribbon copy, not carbons, clearly typed and double-spaced throughout, including tables, footnotes, and bibliographies. (Note -- only one copy is needed.) Footnotes should be typed at the bottom of the page.

(3) ABSTRACTS are requested for all except very short articles.

(4) CAUSES OF DISEASES should be named. For bacteria, fungi, nematodes, etc., give the Latin name of the organism; for viruses either or both the accepted common name of the virus or a Latin name if you prefer it and there is one; for non-parasitic diseases state the causal factor if it is known. If the cause of a disease has not been determined say so.

(5) LITERATURE REFERENCES should be given in alphabetical order and numbered for citation in the text. We follow the AIBS suggestion of placing the year of publication after the author's name. Please check your references carefully since we cannot do it for you. Be sure that text citations and bibliography agree; that foreign-language references are correct; that number or month is cited for periodicals that are not paged consecutively throughout the volume.

(6) NAMES OF FUNGICIDES should be given according to the suggestion of McCallan et al. in Phytopathology (45 (6): 295-302. 1955).

(7) ILLUSTRATIONS should be sent to us unmounted. To prevent mistakes, write figure numbers on the back, and mark the top of each print when necessary. A sketch can show a preferred arrangement but please keep in mind page size, shape, and standard reduction (see above under General), and remember that figure titles and legends are part of the page. Lettering should be clear and large enough to be legible after reducing. Drawings, maps and graphs can be photographs or originals, but should be finished and ready for reproduction, not just sketches.

(8) TABLES should be carefully thought out with particular attention to the Reporter's limitations in reproduction. Make titles and headings definite and self-explanatory. Designate footnotes in tables with superscript lower-case letters. Be sure that text discussion agrees with the data in the table. Do not abbreviate names of crop varieties.

(9) REPRINTS cannot be supplied since there is no way in which we can be reimbursed. However,

(10) The MULTILITH PLATES from which reprints can be made will be sent if requested at the time the article is submitted. The press size of these plates used for the Reporter is designated as small -- maximum image 9 1/2 by 13 inches, maximum paper size 9 3/4 by 14 inches -- for Model 1250. Most of the Experiment Stations have this type of multilith machine.

ACCEPTANCE OF MANUSCRIPTS

The increase in the volume of pertinent material offered for publication in the Plant Disease Reporter has made it necessary to limit the subject matter and the length of articles accepted. The subject matter should emphasize new things in plant pathology, such as new records of disease occurrence, serious outbreaks and epidemics, conditions affecting development of plant diseases, techniques of investigation including instrumentation, new discoveries in control including new materials and their evaluation. Manuscripts will be limited to 12-double-spaced typed pages, including tables, graphs, and photographs. Because of reproduction costs photographs should be kept to a minimum. Insofar as possible, material should be presented as graphs rather than tables. Papers cannot be accepted for publication that report routine control experiments, reviews, bibliographies without annotation, results of routine surveys, mere summaries or lists of plant diseases. By following this procedure we hope to continue publishing all articles promptly.

Paul R. Miller

Manuscripts for and correspondence about this publication should be sent to:

PLANT DISEASE REPORTER
Mycology and Plant Disease Reporting Section
Crops Protection Research Branch
Plant Industry Station
Beltsville, Maryland

THE PLANT DISEASE REPORTER

Crops Research Division — Plant Industry Station, Beltsville, Maryland
Volume 43 — December 15, 1959 — Number 12

CONTENTS

1. Pea streaks naturally incited by combinations of viruses
W. T. SCHROEDER, et al. 1219

2. The Cronartium coleosporioides complex in the black hills
ROGER S. PETERSON 1227

3. Avocado diseases in Latin America
GEORGE A. ZENTMYER 1229

4. Polygonum persicaria L., a new host for Heterodera trifolii Goffart
R. MANKAU 1230

5. Control of root-knot nematodes on vegetables on the Eastern Shore of Maryland with 1,2-dibromo-3-chloropropane-fertilizer mixtures
J. G. KANTZES, et al. 1231

6. Decay of poinsettia cuttings by the soft rot bacterium, Erwinia carotovora (Jones) Holland
MARLIN N. ROGERS 1236

7. Occurrence of southern corn rust in Wisconsin
M. S. PAVGI and A. L. FLANGAS 1239

8. Helminthosporium rostratum Drechs. on rice in India
S. B. CHATTOPADHYAY and C. DASGUPTA 1241

9. An outbreak of phloem necrosis in Alabama
E. A. CURL, et al. 1245

10. An improved method of testing the tolerance of strawberry varieties and new selected seedlings to virus infection
P. W. MILLER 1247

11. Effect of storage time and temperature on control of seed-borne safflower rust by volatile mercury fungicides and the efficacy of certain seed protectants and antibiotics
C. A. THOMAS 1250

12. Relative resistance of some strawberry varieties, species, selections and seedling populations to powdery mildew at Agassiz and Abbotsford, British Columbia
HUGH A. DAUBENY 1253

13. 1959 wheat streak mosaic epiphytotic in Kansas
CLAUDE L. KING and WEBSTER H. SILL, Jr. 1256

14. Host-parasite relationships of various plants and the sugar-beet nematode (Heterodera schachtii)
A. MORGAN GOLDEN and THELMA SHAFER 1258

15. Comparative treatments for powdery mildew of apple
L. E. DICKENS and W. J. HENDERSON 1263

16. Summary of plant diseases in Virginia, 1959
S. B. FENNE 1264

17. The control of ergot in cereal crops
W. P. CAMPBELL and H. A. FREISEN 1266

18. Reaction of rescuegrass varieties and strains to the head smut fungus
HOWARD W. JOHNSON 1268

19. Additional unrecorded hosts of Armillaria mellea
ROBERT D. RAABE 1270

20. Relation of crop sequence to tassel smut of corn
ROY D. WILCOXSON and R. P. COVEY 1271

21. Reducing Amaryllis leaf spot by spraying
D. L. GILL 1272

22. Effects of soaking Easter lily bulbs in Puratized Agricultural Spray or demeton, or in combined soaks, on aphids, black scale and yield
D. L. GILL 1274

23. Symptoms of cachexia in Orlando tangelo, none in sweet lime and false symptoms associated with purple scale infestations
T. J. GRANT, et al. 1277

24. Reaction of cabbage varieties and clubroot-resistant lines to root-knot nematodes
N. N. WINSTEAD 1280

25. A study of Erysiphe graminis races on barley in West Virginia
S. C. SHIH, et al. 1282

26. Nitrogen fertilization on the response of soybeans infected with Heterodera glycines
J. P. ROSS 1284

27. Survival of loose smut in barley seed
KARL D. FEZER 1287

28. Crown rust on barley in Minnesota
R. W. LUTEY and R. P. COVEY 1287

29. Oak wilt in Oklahoma and Arkansas
A. F. VERRALL, et al. 1288

30. Corrections 1288

PEA STREAKS NATURALLY INCITED BY COMBINATIONS OF VIRUSES[1]

W. T. Schroeder, R. Provvidenti and F. L. McEwen

Abstract

Pea "streaks" occurring naturally in New York State, in many instances indistinguishable from those ascribed to single viruses, were demonstrated by a process of analysis through differential hosts and synthesis through recombination of isolated viruses to be caused by a complex of the red clover vein mosaic virus and strains of the bean yellow mosaic virus. Streaks of this nature are conjectured to be of more importance than heretofore considered because of the wide distribution of the component viruses throughout the pea-growing areas. One of the mosaic component viruses isolated, closely resembling bean yellow mosaic virus if not a strain thereof, is infectious to varieties of pea normally resistant to bean yellow mosaic virus. In combination with the red clover vein mosaic virus it produced a severe streak on varieties of pea such as W. R. Perfection and Bonneville.

INTRODUCTION

In 1958, outbreaks of severe "streak" occurred in a number of commercial pea fields in western New York. A 20-acre field of Giant Stride grown in rows at Auburn, New York for fresh market peas was plowed under without a pod having been picked, because over 95 percent of the plants were damaged by the disease. A field of Thomas Laxton freezing peas at Bergen, New York was materially reduced in yield by the "streak." Other instances of "streak" were reported in 1958 and in 1959 from commercial pea plantings. A considerable amount was observed in breeding plots near Geneva, New York. Although usually predominant, pea enation mosaic occurred only sparingly in the above years.

Streak diseases of pea are not new to New York, having been observed before 1958. Their importance in prior years, however, was minimized, probably because they were confounded or masked by the pea enation mosaic syndrome. Furthermore, attempts to transmit the "streak" viruses from pea to pea usually failed. The few successful inoculations involved viruses capable of producing streaks singly, such as alfalfa mosaic and cucumber mosaic viruses.

The purpose of this paper is to present evidence that certain of the so-called "streaks," as found in New York, result from combinations of specific viruses that occur in nature, either singly or together in some reservoir hosts. Previous unsuccessful attempts at transfer were probably due to the fact that the host pea variety usually used is resistant to certain strains of one of the viruses in the combination.

STREAK SYNDROME ON PEA

Symptoms of the various "streak" specimens collected during 1958 and 1959 were similar in many respects, often identical, to those of streaks attributed by other investigators to single viruses (2, 7, 16). In the field the most striking symptoms occurred on the pods, and ranged from a brown, roughened and/or pitted condition on filled or partially filled pods to purple-brown, completely flat pods. Streaks of similar color on the stems and peduncles preceded pod formation and remained as prominent symptoms. Vascular discoloration often accompanied the stem streaking. Erratic symptoms that appeared were a slight mottle, a faint indication of vein clearing, axillary bud proliferation and a yellowing of the terminal foliage, with or without veinal necrosis or necrotic flecking (Fig. 1. A, B, C).

In the greenhouse, at a temperature range of 80° to 85° F mechanical inoculations of pea suscepts, such as Perfected Wales, with juice from plants exhibiting the above symptoms usually produced a faint chlorosis of the leaves immediately above those inoculated within 6 to 8 days, followed closely by a greyish-tan streaking and slight epinasty of the terminal growth. Sometimes a mild rosetting appeared at the terminal growth. Leaves later became progressively chlorotic with slight veinal necrosis, and ultimately wilted and died (Fig. 2). At a constant greenhouse temperature range of 60° to 65° some of the aphid-inoculated pea suscepts developed symptoms similar to those observed on field-grown plants.

[1] Approved by Director of the New York State Agricultural Experiment Station, Geneva, New York as Journal Paper No. 1194.

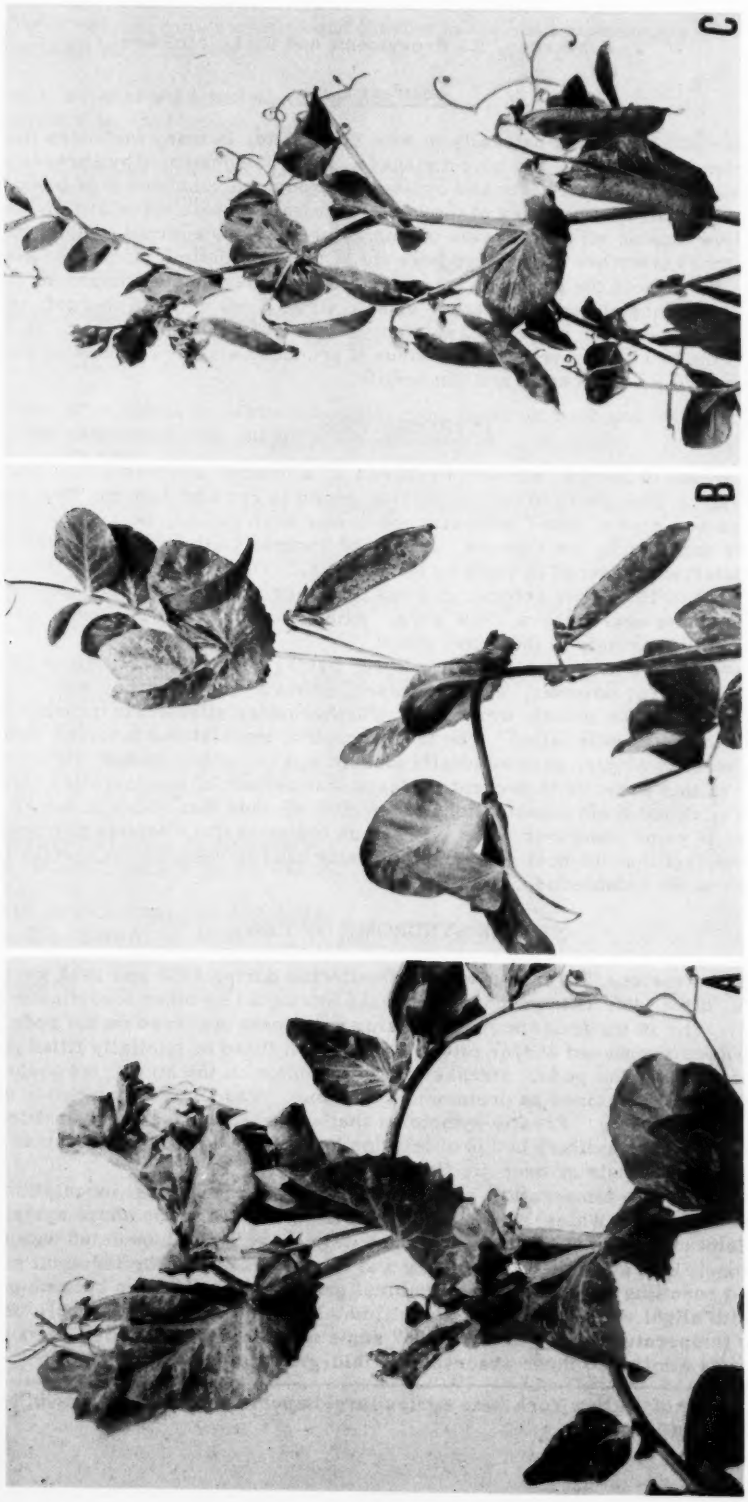


FIGURE 1. Naturally occurring pea "streaks" resulting from combinations of viruses. A -- Giant Stride infected with RCVMV and BV2, 1958 (58-2). B -- Thomas Laxton infected with RCVMV and BV2, 1959. C -- Breeding Line 201 infected with RCVMV and PV2, 1958 (58-1).

FIGURE 2. Greenhouse "streak" on Perfected Wales resulting from aphid transfer of sap from a synthesized "streak" plant of the same variety. Greenhouse temperature range of 80° to 85° F.

ANALYSES OF THE STREAK VIROSES

The complex natures of the "streak" viroses were discovered only after 25 pea accessions, obtained from the Northeast Regional Plant Introduction Station at Geneva, New York, were evaluated for resistance against two of the "streak" isolates, 58-1 and 58-2 (Fig. 1, A and C). Of the accessions tested against 58-2, 11 were susceptible and exhibited typical "greenhouse" streak symptoms, 7 appeared to be heterozygous and 7 showed no streak symptoms and were classed as resistant. Among those accessions designated as susceptible, an occasional plant showed no streak but appeared to be infected with the yellow bean mosaic virus (BV2). Discrete symptoms of BV2 never appeared in plants of the lines classed as resistant, but well-defined symptoms of Wisconsin pea stunt, caused by the red clover vein-mosaic virus or RCVMV (5), later developed in a large proportion of the plants. These data suggested that the "streak" of 58-2 isolate might be the result of a combination of these two viruses. Attempts were therefore made to separate the components of the original isolate by passing it through the differential hosts, Red Kidney bean (susceptible to BV2 but resistant to RCVMV) and Bonneville pea (resistant to BV2 but susceptible to RCVMV). Only yellow bean mosaic appeared on Red Kidney bean and only Wisconsin pea stunt developed on Bonneville. Mechanical inoculations with juice from each of the above infected differential hosts failed to produce any "streak" on Perfected Wales. While not conclusive, this evidence suggested that the streak from isolate 58-2 resulted from the combination of BV2 and RCVMV.

Resistance to 58-1 among the 25 pea accessions was essentially the same as that obtained with 58-2, but isolate 58-1 produced somewhat different symptoms on the differential hosts. The "streak" symptoms were identical, but the mosaic observed on some plants of the streak-susceptible lines resembled the "speckle" strain of common pea mosaic virus, PV2B, described by Stubbs (15). Unlike 58-2, this isolate was not infectious to Red Kidney bean. On white lupin (Lupinus albus), however, 58-1 produced a foliar mottle and distortion, followed by stunting of the plant in the greenhouse at 80° to 85°F, symptoms similar to those described on that host by Chamberlain (1) for common pea mosaic. Under the same conditions the mosaic component of 58-2 produced a bud necrosis on white lupin, followed by the death of the plant. Primary inoculations of white lupin with the mosaic component of 58-1 (presumably PV2 or a strain thereof), followed later at the foliar mottle stage by inoculation with BV2, failed to produce bud necrosis and plant death at a greenhouse temperature of 80° to 85° F. This indicated cross-protection and a close relation between the two mosaic components. Goodchild (3) previously proved the relationship of BV2 and PV2 by serology and a somewhat different cross-protection test.

Continued investigations confirmed the value of Red Kidney bean, white lupin and Bonneville pea as differential hosts for the analysis of "streak" resulting from combinations of RCVMV with either BV2 or PV2. Red Kidney bean and white lupin were insusceptible in these tests to RCVMV and identified the mosaics; whereas, Bonneville pea, being resistant to both mosaics,

identified RCVMV. Red Kidney bean also served to differentiate alfalfa mosaic virus from the above complexes by virtue of the local lesion symptom. Tobacco (Nicotiana rustica), which also develops local lesions followed by systemic infection when inoculated with alfalfa mosaic virus, was included among the differential hosts. National Pickling cucumber (Cucumis sativus) screened out the cucumber mosaic viruses from the above complex.

Numerous subsequent analyses of "streaks" in 1958 and 1959 with the above differential hosts indicated a common and widespread occurrence of the combination "streaks" in peas in New York. For example, in 1959, of 22 streak specimens collected from widely scattered pea-growing areas in western New York, 12 yielded BV2 and RCVMV, 6 yielded PV2 and RCVMV, 2 yielded alfalfa mosaic viruses, 1 produced a slight mottle only on Perfected Wales, and 1 was negative. The causal complex of the above-described "streaks" of pea were also found in combination within natural reservoir hosts such as red and alsike clovers.

SYNTHESIS OF STREAKS

By analysis, it was determined that the New York "streaks" contained RCVMV with either PV2 or BV2. That evidence however was insufficient to prove that the causal entities of "streak" were RCVMV and either BV2 or PV2, since the possibility existed that these components may have occurred coincidentally with a third virus, the real cause. To eliminate the possibility of a third virus as the cause, it was deemed advisable to attempt the synthesis of "streaks" by combining RCVMV with BV2 or PV2. The isolates of RCVMV and BV2 used had been obtained previously from D. J. Hagedorn, University of Wisconsin. The PV2 culture, identified as such on the basis of symptoms on Perfected Wales and white lupin, was isolated from one of the breeding lines in the pea nursery. Separate juice extracts, each containing one of the component viruses, were combined in equal amounts and used with carborundum to mechanically inoculate plants of either Perfected Wales or Giant Stride. In every instance both leaves of plants at the two-leaf stage were inoculated with the virus mixtures and the inoculated plants were maintained in the greenhouse at a temperature range of 80° to 85° F.

Pea "streak" evolved in Perfected Wales plants from a combination of RCVMV and either BV2 or PV2 in the following manner. Within 5 to 6 days after inoculation a slight vein clearing appeared on the fourth leaf, while the third leaf remained free of any symptoms. Mottle that was characteristic of BV2 or PV2, depending upon which virus was used, followed on the fifth leaf. Control plants inoculated with BV2 or PV2 alone showed a similar sequence of symptoms. About 14 days after inoculation symptoms of Wisconsin pea stunt appeared on plants inoculated with the virus mixture and on the control plants inoculated solely with RCVMV. One to 2 days later, a streaking and collapse of the stems occurred 2 to 3 internodes above the point of inoculation on plants that received the virus mixture. No such streaking developed on control plants inoculated with the individual viruses. Tissue from the "streaked" plants was triturated and another group of Perfected Wales plants was inoculated with the extracted juice. The sequence of symptoms described above for the original transmission were not as well defined in the second serial transfer of the virus complex. In some instances only the slight mottle on the fourth leaf preceded the development of the streak phase. With the third serial transfer, the combination of the viruses seemed to be well fixed and, as a result, the slight mottle on the fourth leaf followed immediately by streak was characteristic. Typical streak under the above greenhouse conditions usually developed completely within 10 days after inoculation with the fixed virus combination. This is an incubation period intermediate to those of the individual viruses involved; namely, BV2 or PV2 and RCVMV, which require 5 to 6 and 13 to 15 days respectively for symptom expression under the same temperature conditions. These results demonstrated that a naturally occurring pea "streak", transmitted to greenhouse plants by mechanical inoculations, could also be synthesized there by combining the two viruses previously determined by analysis through differential hosts to be the causal entities. A third virus was not involved. When Giant Stride was used as the host variety of pea in the synthesis, using RCVMV and either BV2 or PV2, typical "greenhouse" streak usually appeared in the original transmission, without the sequence of intermediate symptoms observed on Perfected Wales.

In 1959 analysis of a "streak"-affected pea plant revealed a virus combination somewhat different from that already described; one which could become very important. RCVMV was isolated as one of the components. The other was a mosaic virus similar to BV2 on Perfected Wales and on Red Kidney bean, but it infected the two pea varieties Bonneville and Perfection, which are generally considered resistant to the bean yellow mosaic viruses. A mechanical mixture of this virus (14B) and RCVMV produced a typical streak on Bonneville in a manner similar to that described for the RCVMV-BV2 combination (Fig. 3). The identity of the 14B virus is currently under investigation.

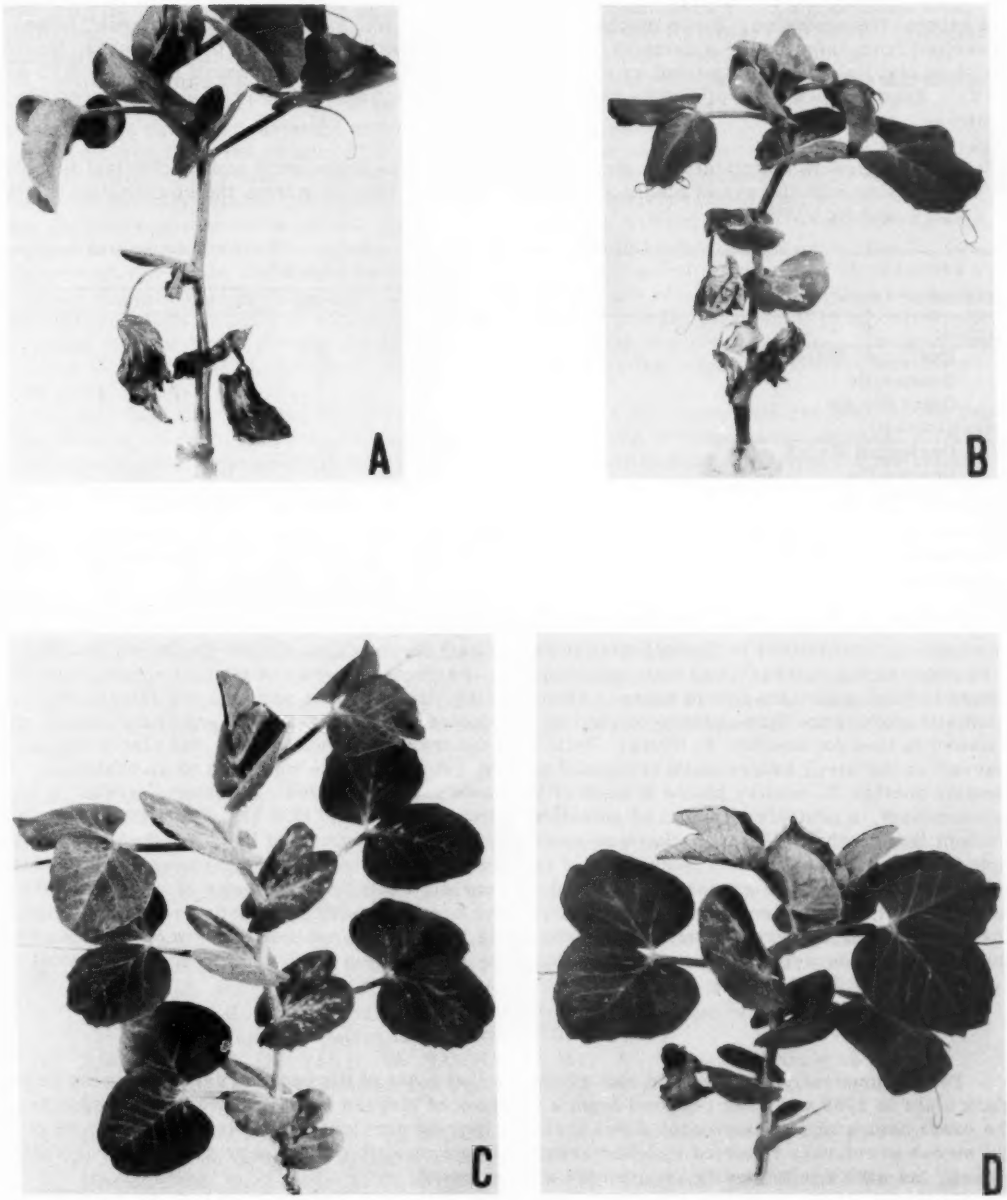


FIGURE 3. Similarity in symptoms between a synthesized "streak" and that incited by alfalfa mosaic virus 575 on Bonneville pea. A -- AMV 575. B -- RCVMV plus 14B, a BV2-like virus. C -- 14B alone. D -- RCVMV. Greenhouse temperature range of 80° to 85° F.

APHID TRANSMISSION OF STREAK

The prevalence of "streaks" in the field during 1958 and 1959 suggested an efficient means of natural transmission. Since mechanical transmission had established that "streak" in pea resulted from infection by a complex of RCVMV and either BV2 or PV2, the pea aphid, Macrosiphum pisi Kalt., was suspected as a vector because it is known to transmit RCVMV, BV2 and PV2. Experiments were therefore designed to determine whether the pea aphid vectored the "streak" complex.

Table 1. Symptom distribution in three varieties resulting from aphid and mechanical inoculation with the sap of plants affected by "streak" resulting from the combination of PV2 and RCVMV.

Method of transmission	Number of plants[a]				
	Inoculated	Healthy	Streak	Mosaic	Stunt
Aphid:					
Perfected Wales	30	20	6	4	0
Bonneville	30	18	0	0	12
Giant Stride	30	15	10	3	2
Mechanical:					
Perfected Wales	30	2	25	3	0
Bonneville	30	4	0	0	26
Giant Stride	30	0	30	0	0

[a]Remained as mosaic -- did not develop streak symptoms for the 2 to 3 months duration of the experiment.

The virus complex was obtained by mixing RCVMV and PV2 and making serial transfers by mechanical inoculations in the following order: Giant Stride-Giant Stride-Perfected Wales. The combination "streak" was well established on Perfected Wales and healthy aphids were allowed to feed upon them for 24 hours. After feeding, three aphids were placed on each of 30 plants in each of the three healthy varieties, Perfected Wales, Bonneville and Giant Stride, and allowed to feed for another 24 hours. Following the transfer of the aphids, the plants which served as the virus source were triturated and the extracted juice was used to mechanically inoculate another 30 healthy plants in each of the same three varieties. Although "streak" was predominant, a scattered amount of mosaic and stunt appeared (Table 1). From these data it is evident that mechanical inoculation was more effective in transmitting "streak" than was the pea aphid. Since the pea variety Bonneville is resistant to PV2, no streak symptoms were produced on this host. However, numerous plants of this variety exhibited symptoms of Wisconsin pea stunt. The transmissions of the individual viruses occurred with both methods of inoculation, the frequency being perhaps greater with the aphid transfers. Another experiment demonstrated that single aphids were capable of transmitting the combination of viruses to produce typical "streak" on Perfected Wales.

DISCUSSION

The demonstration by analysis and synthesis that most of the pea "streak" occurring in New York State in 1958 and 1959 resulted from a complex of viruses leads to speculation regarding the exact nature of streak viroses elsewhere. It may be pure conjecture to attribute much of the streak previously reported in other areas to infection with the same or similar virus complexes, but such conjecture is not entirely without basis.

On the basis of symptoms, New York pea "streaks" are indistinguishable from many of the streak viroses ascribed in the literature to single viruses. Strains of the yellow bean mosaic virus, among which PV2 should be included, are widely distributed in pea-growing areas from New York to the Far West (4, 8, 9, 11). The red clover vein mosaic virus, which occurs commonly in Wisconsin (6), has frequently been observed and repeatedly recovered in New York pea fields. Thus, in many pea-growing areas ample opportunity exists for complex infections with BV2 and RCVMV that result in the production of "streak." For example, the pea streak in Minnesota which Oshima and Kernkamp (12) transmitted from red clover to Early Perfection may have resulted from the combination of the pea mosaic and pea stunt viruses which they also trans-

mitted from red clover to Early Perfection.

Other instances of the effects of combinations of pea viruses have been reported. Pierce (13) was probably the first to synthesize a "greenhouse" streak similar to that reported in this paper when he inoculated peas with an artificial mixture of bean virus 2 and a broadbean local lesion virus, both of which, however, had been obtained previously from red clover. McWhorter (10), as a result of surveys conducted in Oregon and Washington, concluded that many of the wilts, yellow tops and tip blights of pea resulted from the interaction of various viruses with the pea enation mosaic virus. It will be interesting to observe what effect the introduction of pea enation mosaic resistant varieties will have on those complexes (14).

It is obvious from the results of these studies that an accurate analysis of "streak" requires the use of a pea variety susceptible to both components of a double-virus streak as well as to viruses individually capable of producing streak. If a pea variety is resistant to one of the viruses causing a double-virus streak, true streak symptoms will not develop on inoculated plants. Resultant failures to reproduce streak may be misinterpreted as disease escapes, and the development of symptoms characteristic of only one of the component viruses may be regarded as the result of accidental contamination. The comparatively few streak transmissions caused by single viruses such as those of alfalfa mosaic or cucumber mosaic may result in an erroneous conclusion that the latter viruses are the sole cause of streak in a given locality. The true double-virus nature of streak, however, would be confounded by the negative transfers and the so-called contaminants.

The fact that most canning and freezing varieties of pea are reportedly resistant to the bean yellow mosaic virus strains does not minimize the importance of combination streaks. The red clover vein mosaic virus actually may be the more important component, since it does produce under some conditions a slight suggestion of a stem streaking. Perhaps the bean yellow mosaic virus merely accentuates that tendency, and other mosaic type viruses may do the same. Furthermore, as pointed out, there already exist certain mosaics of pea, whether they be strains of bean yellow mosaic or not, that are capable of causing mosaic on the resistant Perfection types and which in combination with the red clover vein mosaic virus can produce "streak" in severe form on pea varieties resistant to the bean yellow mosaic virus. The discovery of resistance in pea to the red clover vein mosaic virus (RCVMV) might prove a boon to the control of many "pea viroses." A search for such resistance is in progress.

The results obtained from these studies point to the urgent need for a standardized procedure in identifying pea viroses. It should be sufficiently detailed to avoid the description of new viruses on the basis of small differences in host range, physical properties, symptoms, and so forth.

Literature Cited

1. CHAMBERLAIN, E. E. 1936. Pea mosaic. Host range and methods of transmission. New Zealand J. Sci. and Tech. 18: 544-556.
2. CHAMBERLAIN, E. E. 1939. Pea streak (Pisum virus 3). New Zealand J. Sci. and Tech. 20: 365A-381A.
3. GOODCHILD, D. J. 1956. Relationships of legume viruses in Australia. Serological relationships of bean yellow mosaic virus and pea mosaic virus. Aust. J. Biol. Sci. : 231-237.
4. GRAVES, C. H. 1953. Incidence of red clover vein mosaic virus in Wisconsin clovers. (Abst.) Phytopathology 43: 473.
5. HAGEDORN, D. J., and E. W. HANSON. 1951. A comparative study of the viruses causing Wisconsin pea stunt and red clover vein mosaic. Phytopathology 41: 813-819.
6. HAGEDORN, D. J., and J. C. WALKER. 1949. Wisconsin pea stunt, a newly described disease. J. Agr. Research 78: 617-626.
7. HAGEDORN, D. J., and J. C. WALKER. 1949. Wisconsin pea streak. Phytopathology 39: 837-847.
8. HAGEDORN, D. J., and J. C. WALKER. 1950. The relation of bean virus 2 to pea mosaic in Wisconsin. Phytopathology 40: 684-698.
9. McWHORTER, FRANK P. 1949. Susceptibility of selections of Perfection peas to strains of yellow bean mosaic. Plant Disease Reptr. 33: 139-144.
10. McWHORTER, FRANK P. 1954. The virus disease complex in canning peas. Plant Disease Reptr. 38: 453-457.
11. McWHORTER, FRANK P., and JOHN HARDISON. 1949. Record and diagnosis

of subclover severely injured by yellow bean mosaic virus. Plant Disease Reptr. 33: 86-88.

12. OSHIMA, NAGAYOSHI, and M. F. KERNKAMP. 1957. Effects of viruses on overwintering of red clover in Minnesota. Plant Disease Reptr. 41: 10.
13. PIERCE, W. H. 1935. The identification of certain viruses affecting leguminous plants. J. Agr. Research 51: 1017-1039.
14. SCHROEDER, W. T., and D. W. BARTON. 1958. The nature and inheritance of resistance to the pea enation mosaic virus in garden pea, Pisum sativum L. Phytopathology 48: 628-632.
15. STUBBS, MERL W. 1937. Certain viroses of the garden pea, Pisum sativum. Phytopathology 27: 242-266.
16. ZAUMEYER, W. J. 1938. A streak disease of peas and its relation to several strains of alfalfa mosaic virus. J. Agr. Research 56: 747-772.

NEW YORK STATE AGRICULTURAL EXPERIMENT STATION, GENEVA, NEW YORK

THE CRONARTIUM COLEOSPORIOIDES COMPLEX IN THE BLACK HILLS

Roger S. Peterson[1]

Abstract

Rust damage to pine in the Black Hills caused by Cronartium coleosporioides (Peridermium harknessii and P. filamentosum) is described for the first time. New host records for the telial stage on Scrophulariaceae are reported.

Cronartium coleosporioides Arth. includes several distinguishable rust parasites of pine stems (2, 10) and their indistinguishable dikaryotic stages on Indian paintbrushes, owl's-clover, and other members of the tribe Euphrasieae (Scrophulariaceae). The present note describes for the first time the occurrence of these forest pests in SouthDakota and eastern Wyoming.

Members of the complex infecting Black Hills pine are western gall rust (Peridermium harknessii Moore) and limb rust (P. filamentosum Peck). P. stalactiforme Arth. & Kern, although it occurs both to the east and west of the Great Plains (1, 7), was not found during this study[2].

The gall-rust attack on Black Hills ponderosa pine is unusually damaging. The ratio of bole infections to branch infections is very much higher in the Black Hills than in outbreaks on the same host species in northern Idaho and western Montana. In open stands on the Hills' extensive burned areas, main-stem cankers are abundant and will cause significant cull when these stands reach merchantable size. In commercial operations, cankered sections of trunks are generally cut out and left in the woods. Often the largest trees in a stand appear to be most severely cankered. Natural and artificial seedings in the large burn near Roubaix, South Dakota were sampled by regularly spaced line counts in the 40- to 60-year-old stands. In the eighteen 100-tree samples the percentage of dominants and codominants having cankers below the 32-foot height varied from 4 to 10, with an average of 7 percent. In two selected straight-line transects in the Windy Flats area, the percentages of potential crop trees cankered were 35 and 47. Nearly all trees in the area bore galls (often numbering hundreds per tree), which have killed branches and adversely affected tree form.

P. harknessii is also abundant on lodgepole pine (Pinus contorta) near Nahant, South Dakota, and on planted Scots pine (P. sylvestris) near Roubaix, South Dakota.

Peridermium filamentosum is not now a damaging pest in the Black Hills. Its presence in South Dakota is of interest mainly as a range extension for the fungus, previously known only as far northeast as north-central Colorado. (So far as the writer can determine, eastern reports of this rust refer actually to P. stalactiforme or Cronartium comptoniae.) P. filamentosum collections from ponderosa pine near Roubaix and Lead, South Dakota were surprising in two respects: (a) pycnidia occurred regularly together with the sporulating aecia (in early July), a phenomenon seldom reported (4), and (b) infections on 10- to 12-year-old pines were not uncommon, although the parasite is known principally from older trees (6). Branch killing by the fungus occurs in the area, but tree mortality was not apparent.

Both of the stem rusts discussed here have been reported to infect Castilleja (Indian paintbrush) and other genera of Euphrasieae, and both have also been reported to be short-cycling, pine-to-pine rusts (2, 3, 9, 10). It is therefore pertinent to study the uredinial and telial stages in the Black Hills, where P. stalactiforme, the known long-cycling rust of this complex, is uncommon or lacking. Cronartium coleosporioides was abundant on herbaceous hosts in early July 1959 throughout the eastern Black Hills, and uncommon but present farther west. Collections were made in South Dakota on:

(a) Castilleja sulphurea Rydb., the known Black Hills host (8);
(b) C. sessiliflora Pursh, a new host record; and
(c) Orthocarpus luteus Nutt., previously collected as a host in South Dakota by G. G. Hedgcock (FP 38754 in the National Fungus Collections), but this State record was not published.

[1] Plant Pathologist, Rocky Mountain Forest and Range Experiment Station, Forest Service, United States Department of Agriculture, with headquarters at Colorado State University in Fort Collins.

[2] Two South Dakota specimens borrowed through the cooperation of Mr. J. A. Stevenson from the National Fungus Collections and labeled "Cronartium stalactiforme" on examination proved to be Cronartium comandrae Peck. This rust has long been known in the Black Hills (5), and several old cankers (but no new infections) were found during the present field study.

Specimens are deposited in the herbarium of the Fort Collins Forest Disease Laboratory.

Uredinia and telia on Castilleja were found close to both P. harknessii and P. filamentosum on pine. In some instances the probability of alternation by the rusts between pine and paintbrush seemed high. However, field evidence of host alternation was not convincing, and inoculation experiments are required to determine the life cycles of these fungi in the Black Hills.

Literature Cited

1. ANDERSON, N. A. 1959. Stalactiforme-rust canker on jack pine in Minnesota. Forest Science. (In press)
2. ARTHUR, J. C. 1934. Manual of the rusts in the United States and Canada. Purdue Research Foundation, Lafayette, Indiana. 438 pp.
3. BOYCE, J. S. 1943. Host relations and distribution of conifer rusts in the United States and Canada. Trans. Connecticut Acad. Arts Sci. 35: 329-482.
4. HAWKSWORTH, F. G. 1953. The pycnial stage of Cronartium filamentosum. Plant Disease Reptr. 37: 246.
5. LUCKENBILL, L. 1935. The pine-comandra rust in the Black Hills. Forest Serv. Region 2, Harney National Forest, The Bulletin 18: 13-14.
6. MIELKE, J. L. 1952. The rust fungus Cronartium filamentosum in Rocky Mountain ponderosa pine. J. Forestry 50: 365-373.
7. MIELKE, J. L. 1956. The rust fungus (Cronartium stalactiforme) in lodgepole pine. J. Forestry 54: 518-521.
8. THURSTON, H. W., Jr., and F. D. KERN. 1931. Notes on some rust collections from Colorado, Wyoming, and South Dakota. Mycologia 23: 77-82.
9. U. S. FOREST SERVICE. 1959. Highlights in research; annual report -- 1958. Intermountain Forest and Range Exp. Sta.
10. WAGENER, W. W. 1958. Infection tests with two rusts of Jeffrey pine. Plant Disease Reptr. 42: 888-892.

ROCKY MOUNTAIN FOREST AND RANGE EXPERIMENT STATION, FOREST SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, FORT COLLINS, COLORADO

AVOCADO DISEASES IN LATIN AMERICA

George A. Zentmyer

During exploration trips in 1956, 1958, and 1959 in search of resistance to Phytophthora root rot of avocado, a number of observations were made of avocado diseases in various countries in Latin America. Root cultures were made to confirm diagnosis of occurrence of Phytophthora cinnamomi, using corn meal agar for isolation of the fungus as described previously (1, 2).

The following diseases were identified on avocado (Persea americana), with general locations:

PHYTOPHTHORA ROOT ROT (causal fungus: Phytophthora cinnamomi)
Mexico: Ciudad Victoria (Tamps.); Atlixco (Puebla); Jungapeo (Michoacan)
Guatemala: San Pedro Carcha (Alta Vera Paz) (isolation from Persea schiedeana)
Honduras: Zamorano (Escuela Agricola Panamericana)
Costa Rica: San Mateo, Orotina
Chile: La Cruz, San Fernando, Malloa
Peru: Chanchamayo Valley
Brazil: Campinas (São Paulo)
Trinidad: Port of Spain
Cuba: Santiago de las Vegas
Puerto Rico: Isabela, Jajuja, Mayaguez

PHYTOPHTHORA TRUNK CANKER (causal fungus: Phytophthora cinnamomi)
Brazil: Piricacaba (São Paulo)

PHYTOPHTHORA BRANCH CANKER (causal fungus: Phytophthora palmivora?)
Honduras: Mt. Uyuca

VERTICILLIUM WILT (causal fungus: Verticillium albo-atrum)
Chile: La Cruz
Ecuador: Guiallabamba Valley

ARMILLARIA ROOT ROT (causal fungus: Armillaria mellea)
Ecuador: Guiallabamba Valley

SUN BLOTCH (virus)
Peru: Chanchamayo Valley

POWDERY MILDEW
Brazil: Campinas

SCAB (causal fungus: Sphaceloma perseae)
Brazil: São Paulo State
Peru: Chanchamayo Valley
Puerto Rico: Isabela

PHYSALOSPORA CANKER (causal fungus: Physalospora perseae)
Peru: La Molina

BOTRYOSPHAERIA CANKER (causal fungus: Botryosphaeria ribis)
Chile: La Cruz

MISTLETOE (Phoradendron sp.)
Mexico: Atlixco (Puebla)

LEAF SPOT (causal fungus: Mycosphaerella perseae)
Mexico: Teziutlan (Puebla)

Literature Cited

1. ZENTMYER, G. A., and W. POPENOE. 1951. Phytophthora cinnamomi on avocado in Honduras. Plant Disease Reptr. 35: 25.
2. ZENTMYER, G. A., J. D. GILPATRICK, and W. A. THORN. 1959. Methods of isolating Phytophthora cinnamomi from soil and host tissue. (Abst.) Phytopathology (in press).

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, RIVERSIDE, CALIFORNIA

POLYGONUM PERSICARIA L., A NEW HOST FOR HETERODERA TRIFOLII GOFFART

R. Mankau[1]

During a study of the development of an Illinois population of Heterodera trifolii on different plants of varying susceptibility, an attempt was made to infest plants which are hosts of other Heterodera species and which had not been previously reported by Gerdemann and Linford (1). The development of this nematode to a very limited extent on two varieties of soybean, a common host of H. glycines Ichinohe, has already been described (3).

An observation of decided interest was noted when rooted cuttings of smartweed (Polygonum persicaria) potted in steamed soil, were each infested with 500 H. trifolii larvae. After 33 days roots were found to bear large numbers of white females filled with eggs and comparable in size to females of the same age on Ladino clover (Trifolium repens), a common host for this nematode. This is a new host for H. trifolii and one that it has in common with the little known species Heterodera weissi Steiner, 1949. H. weissi, according to Steiner (4), is known to occur in numerous States east of the Rocky Mountains and has been found attacking only various species of the genus Polygonum (knotweeds). Jones (2) reported that an occasional cyst of a population of H. schachtii which he studied developed on the roots of Polygonum persicaria.

Larval measurements described for H. weissi range from a length of 320 μ to 388 μ and males apparently are common, whereas lengths of H. trifolii larvae from the population used ranged from 475 μ, to 585 μ, and no males have ever been observed. It would appear relatively easy to separate either of these species occurring on Polygonum on the basis of larval measurements and the occurrence or absence of males.

Literature Cited

1. GERDEMANN, J. W., and M. B. LINFORD. 1953. A cyst-forming nematode attacking clovers in Illinois. Phytopathology 43: 603-607.
2. JONES, F. G. W. 1950. Observations on the beet eelworm and other cyst-forming species of Heterodera. Ann. Appl. Biol. 37: 407-440.
3. MANKAU, (G), R., and M. B. LINFORD. 1956. Soybean varieties tested as hosts of the clover cyst nematode. Plant Disease Reptr. 40: 39-42.
4. STEINER, G. 1949. Plant nematodes the grower should know. Proc. Soil. Sci. Soc. Florida, 1949 (year 1942), 4B, p. 99.

UNIVERSITY OF CALIFORNIA, RIVERSIDE, CALIFORNIA

[1] Assistant Nematologist, Department of Plant Nematology, Citrus Experiment Station, University of California, Riverside, California.

CONTROL OF ROOT-KNOT NEMATODES ON VEGETABLES ON THE EASTERN SHORE OF MARYLAND WITH 1,2-DIBROMO-3-CHLOROPROPANE-FERTILIZER MIXTURES[1]

J. G. Kantzes[2], W. R. Jenkins[2], and R. A. Davis[3]

Abstract

Fertilizer premixed with a granular formulation of 1,2-dibromo-3-chloropropane (DBCP) has given excellent control of root knot of cantaloupe and tomato on the Eastern Shore of Maryland. Cantaloupe seed were planted at the time of application of 500 pounds/acre 5-10-5 fertilizer mixed with both 24 and 35 pounds/acre of DBCP. Tomatoes were treated 1 week after transplanting with either 400 or 500 pounds/acre 5-10-10 fertilizer mixed with 24 pounds/acre DBCP. All treatments resulted in significant reductions in the root-knot index as compared with untreated checks. The practical value of this method of applying DBCP has been illustrated in these trials.

With the introduction of 1,2-dibromo-3-chloropropane (DBCP), a material was made available which could be used for nematode control on established plants (3). Successful control of the root-knot nematode (Meloidogyne hapla) on strawberry was accomplished on the Eastern Shore of Maryland by post-planting treatment of parent stock with DBCP, either in liquid form or adsorbed onto clay granules and premixed with fertilizer (4). This work indicated the possibility of using the nematocide-fertilizer mixture for nematode control on other crops. Moreover, the elimination of special equipment for fumigation would be of great benefit to the farmer.

Laboratory, greenhouse, and field tests indicated that DBCP could be safely used at planting time on a wide range of seeded crops. Further, it was found that tomato seedlings could be treated with a nematocidal level of DBCP with no apparent injury (2). Results reported in this paper are based upon the current year's work; however, the results agree with preliminary investigations conducted during 1957 and 1958.

GENERAL PROCEDURES

All of the test fields were located near Salisbury on the Eastern Shore of Maryland. Soil was identified as a Sassafrass sandy loam. These fields were known to have a high infestation of the root-knot nematode, Meloidogyne incognita acrita Chitwood, 1949.

Prior to field application, the granular DBCP[4] for use in all experiments was premixed with fertilizer at the required rates at a commercial fertilizer plant.

All nematocide-fertilizer treatments were applied either at seeding time or as a post-planting treatment with conventional fertilizer side-dressing equipment attached to a planter or tractor.

At the end of the season plant samples were dug from the different field experiments and the root systems were scored for incidence of root knot, using an index system of 1 to 5 (1 = 0 percent of roots galled; 2 = 1-25 percent; 3 = 26-50 percent; 4 = 51-75 percent; 5 = 76-100 percent infection).

Field experiments for nematode control included one test on the cantaloupe variety Early May and two tests on the tomato variety KC-146.

CANTALOUPES

Materials and Methods: This test field, containing approximately 3 acres, was divided into three blocks, each of which contained three treatments. Cantaloupe seed was planted in rows 5 feet apart and thinned to plants approximately 18 inches apart in the row. Rows were approximately 700 feet in length. Treatments were as follows:

---

[1]Scientific Art. No. A812, Contribution No. 3084 of the Maryland Agricultural Experiment Station, Department of Botany.

[2]Assistant Professor of Plant Pathology.

[3]Graduate Assistant in Plant Pathology.

[4]Supplied as 25 percent technical Nemagon by the Agricultural Chemicals Division, Shell Chemical Corporation, New York, New York.

Treatment 1 -- Fertilizer (5-10-5 at the rate of 500 pounds per acre).
Treatment 2 -- Fertilizer as above premixed with nematocide (25 percent granular DBCP at the rate of 24 pounds per acre).
Treatment 3 -- Fertilizer as above premixed with nematocide (25 percent granular DBCP at the rate of 35 pounds per acre).

All treatments were applied at time of planting with a tractor-pulled conventional seed planter with fertilizer attachment. The fertilizer and nematocide-fertilizer mixtures were applied in bands to a depth of approximately 3 to 4 inches on both sides of the cantaloupe seed and approximately 4 inches from the center.

After the last harvest, 60 plant samples were dug at random from each treatment in two blocks and root systems were scored for incidence of root knot.

Results: Granular DBCP premixed with fertilizer at the rates of 24 and 35 pounds per acre was highly effective in controlling root knot on cantaloupe in this field experiment (Table 1 and Fig. 1). There was no reduction in stand or any evidence of phytotoxicity in the treated areas. Generally, during the latter part of the season vines in the treated area appeared to be more vigorous than those in the untreated area.

Table 1. Control of root knot of cantaloupe caused by the nematode Meloidogyne incognita acrita with 1,2-dibromo-3-chloropropane premixed with fertilizer and applied as a side-dressing at seeding time. Fertilizer applied at the rate of 500 pounds 5-10-5 per acre.

Treatment	Nematocide[a] pounds/acre	Root-knot index[b]
1	None	4.4[c]
2	24	1.4
3	35	1.5

[a]25 percent by weight technical Nemagon.
[b]Indexed as follows: 1 = 0 percent roots galled; 2 = 1-25 percent; 3 = 26-50 percent; 4 = 51-75 percent; 5 = 76-100 percent.
[c]Mean of two samples of 60 plants each.

TOMATOES

Materials and Methods: Experiments on tomatoes were conducted on two different farms which shall be designated as Farm A and Farm B.

The test field on Farm A, about 4 acres in size, was divided into three blocks. Each block consisted of two plots with 12 rows in each plot and rows 5 feet apart. Tomato plants were spaced approximately 24 inches apart in the row. There were two treatments in each of the blocks as follows:

Treatment 1 -- Fertilizer (5-10-10 at the rate of 500 pounds per acre).
Treatment 2 -- Fertilizer as above premixed with nematocide (25 percent granular DBCP at the rate of 24 pounds per acre).

One week after transplanting, treatments were applied with conventional side-dressing equipment. They were applied in bands at a depth of approximately 4 to 5 inches and approximately 8 inches from the center.

At the end of the harvest season 160 plants were dug at random from each of the treatments in two of the blocks. Root systems were scored for incidence of root-knot infection.

On Farm B the experimental field, about 3 acres in size, was divided into three blocks consisting of three plots each, 25 feet wide and approximately 500 feet long. Tomato plants were spaced approximately 24 inches apart in rows 5 feet apart.

There were three treatments replicated and randomized in each of three blocks. The treatments were as follows:

Treatment 1 -- Fertilizer (5-10-10 at the rate of 400 pounds per acre).
Treatment 2 -- Fertilizer as above. Preplant application of D-D[5] mixture at the rate of 20 gallons per acre.
Treatment 3 -- Fertilizer as above premixed with nematocide (25 percent granular DBCP at the rate of 24 pounds per acre).

[5]Supplied by the Agricultural Chemicals Division, Shell Chemical Corporation, New York, New York.

The D-D mixture (dichloropropene and dichloropropane) was applied under pressure with a tractor mounted injection rig 2 weeks prior to planting tomato transplants. Injection chisels were spaced 12 inches apart and the fumigant was placed at a depth of approximately 5 to 6 inches at the broadcast rate of 20 gallons per acre.

Fertilizer and the nematocide-fertilizer mixture were applied to the plots with conventional tractor mounted side-dressing equipment 1 week after transplanting. The materials were applied as bands 3 to 4 inches deep and about 6 inches from the center.

At the end of the season, 60 plant samples were dug at random from each of the replications of each treatment and the root systems scored for incidence of root knot.

Results: Granular DBCP premixed with fertilizer at the rate of 24 pounds per acre was effective in controlling root knot on tomato transplants (Tables 2 and 3). There was no evidence of plant injury following treatment. On Farm B, where D-D mixture was used as a comparison with DBCP, the results of an analysis of the data showed that there was no significant difference in degree of control between treatments. However, both treatments significantly reduced the root-knot index over the untreated plots.

Contrast of root systems of tomato plants at the end of the season from a DBCP-treated plot and from an untreated plot is illustrated in Figure 2.

Table 2. Control of root knot of tomatoes caused by Meloidogyne incognita acrita on Farm A with 1,2-dibromo-3-chloropropane premixed with fertilizer and applied as a side-dressing 1 week after transplanting. Fertilizer applied at the rate of 500 pounds 5-10-10 per acre.

Treatment	Nematocide[a] pounds/acre	Root-knot index[b]
1	None	4.2[c]
2	24	1.3

[a]25 percent by weight technical Nemagon.
[b]Indexed as follows: 1 = 0 percent roots galled; 2 = 1-25 percent; 3 = 26-50 percent; 4 = 51-75 percent; 5 = 76-100 percent.
[c]Mean of two samples of 160 plants each.

Table 3. Control of root knot of tomatoes caused by Meloidogyne incognita acrita on Farm B with 1,2-dibromo-3-chloropropane premixed with fertilizer and applied as a side-dressing 1 week after transplanting compared with a preplant broadcast treatment of D-D mixture. Fertilizer applied at the rate of 400 pounds 5-10-10 acre.

Treatment	Nematocide rate/acre	Root-knot index[a]
1	None	3.8[b]
2	24 pounds DBCP[c]	2.1
3	20 gallons D-D	2.1
LSD .05		1.2

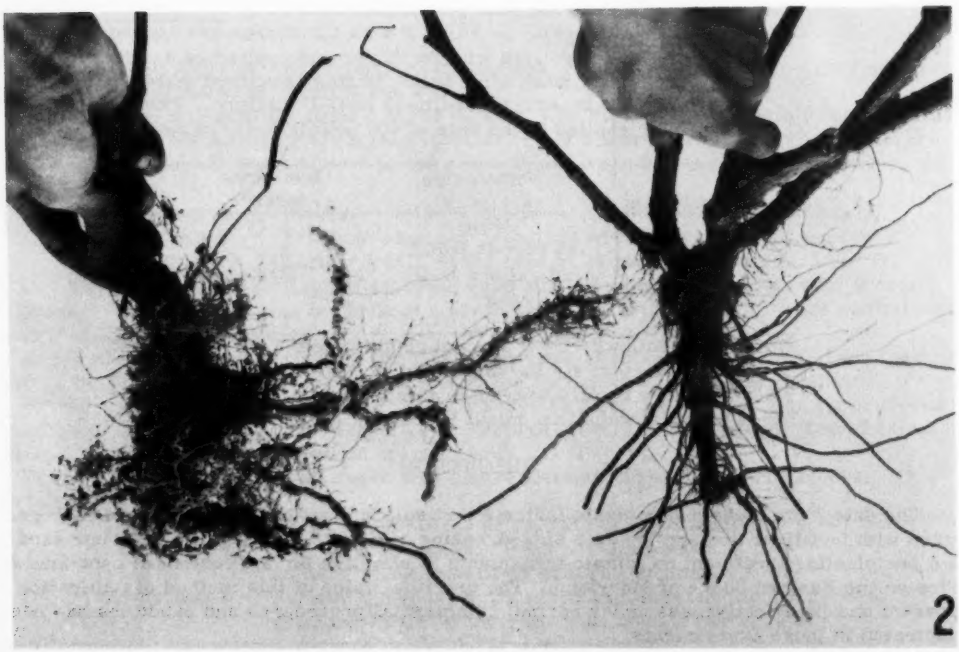
[a]Indexed as follows: 1 = 0 percent roots galled; 2 = 1-25 percent; 3 = 26-50 percent; 4 = 51-75 percent; 5 = 76-100 percent.
[b]Mean of three replications of 60 samples each.
[c]25 percent by weight technical Nemagon.

DISCUSSION

The data from these experiments indicate that soil fumigation with granular DBCP premixed with fertilizer and applied as a side-dressing at time of planting of cantaloupe seed or as a postplanting treatment on tomato transplants is effective for the control of root-knot nematodes on the Eastern Shore of Maryland. The practical value of this method of fumigation is apparent and its effectiveness under normal commercial procedures and practices has been illustrated in these experiments.

FIGURE 1. Control of root knot of cantaloupe with 24 pounds of a granular formulation of 1,2-dibromo-3-chloropropane premixed with 500 pounds 5-10-5 fertilizer per acre on right. Check plant on left treated with 500 pounds 5-10-5 fertilizer only.

FIGURE 2. Control of root knot of tomato with 24 pounds of a granular formulation of 1,2-dibromo-3-chloropropane premixed with 500 pounds 5-10-10 fertilizer per acre on right. Check plant on left treated with 500 pounds 5-10-10 fertilizer only.



In these trials the fertilizer-nematocide mixture was applied at a minimum depth of 3 to 4 inches. A greater depth of fertilizer placement would not be desirable for seeded crops and a more shallow depth of placement of DBCP possibly would not have been effective in root-knot control. This "compromise depth" of fertilizer-nematocide mixture placement gives good control of root knot under the conditions of these experiments. The physical nature of the soil probably allowed for adequate dispersal of the fumigant throughout much of the root zone. Gilpatrick et al. (1) stated that for shallow-rooted crops where nematode control in the top 12 to 18 inches is desired, a shallow placement depth of 2 to 4 inches for DBCP seems adequate. The relatively low volatility of DBCP is unique among fumigants and probably contributes to its greater efficiency when applied at shallow depths. This fact may account for the effectiveness of the granular DBCP-fertilizer mixtures in control of root knot in these experiments.

Literature Cited

1. GILPATRICK, J. D., S. T. ICHIKAWA, M. TURNER, and C. W. McBETH. 1956. The effect of placement depth on the activity of Nemagon. Phytopathology 46: 529-531.
2. KANTZES, J. G., O. D. MORGAN, and W. R. JENKINS. 1957. The possible use of 1,2-dibromo-3-chloropropane ("Nemagon" and "Fumazone") on vegetable crops. Trans. Penin. Hort. Soc. 47: 7-8.
3. McBETH, C. W., and G. B. BERGESON. 1955. 1,2-dibromo-3-chloropropane---a new nematocide. Plant Disease Reptr. 39: 223-225.
4. POTTER, H. S., and O. D. MORGAN. 1956. Nemagon control of root-knot nematode on strawberries. Plant Disease Reptr. 40: 187-189.

DEPARTMENT OF BOTANY, UNIVERSITY OF MARYLAND AGRICULTURAL EXPERIMENT STATION, COLLEGE PARK

DECAY OF POINSETTIA CUTTINGS BY THE SOFT ROT BACTERIUM, ERWINIA CAROTOVORA (JONES) HOLLAND[1]

Marlin N. Rogers[2]

Abstract

A rapid and destructive decay of newly propagated poinsettia cuttings (Euphorbia pulcherrima) under warm, moist greenhouse conditions was found to be caused by the soft rot bacterium, Erwinia carotovora. Inoculation apparently occurred accidentally while cuttings were being prepared for insertion into the rooting medium. Since no earlier citation can be found in the literature, this is believed to be the first report of such an occurrence. Careful sanitation to prevent inoculation of the cuttings is the only practicable control measure that can be recommended.

INTRODUCTION

In July 1959 a large commercial florist in Missouri who specializes in the production of pot plants submitted a group of badly decayed poinsettia cuttings to this laboratory for diagnosis. Five thousand cuttings had been taken from apparently healthy and vigorously growing stock plants, trimmed, dipped in water to stop the flow of milky sap, treated with root promoting hormone powder and inserted in perlite under mist propagation for rooting in accordance with standard commercial practice. Within 24 hours the basal portions of over 90 percent of the stems were completely rotted to a height of 3 to 4 inches. The rot was typically a very watery type of decay and resulted in complete disintegration of the affected portion of the stem. One of the cuttings submitted originally is shown in Figure 1.

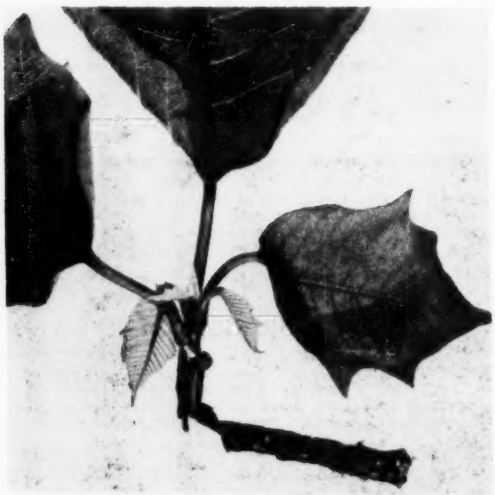


FIGURE 1. One of the rotted cuttings originally submitted, showing complete disintegration of the lower 3 inches of the stem, which had occurred within 48 hours after removal from the stock plant.

IDENTIFICATION OF THE PATHOGEN

Microscopic examination of the rotted tissue disclosed no fungus mycelium, but great quantities of motile bacteria were present. Isolations for bacteria were made directly from the affected tissue by dilution-plate techniques, and also by rubbing portions of the rotted

[1]Contribution from the Missouri Agricultural Experiment Station, Journal Series No. 2080. Approved by Director.

[2]Department of Horticulture, College of Agriculture, University of Missouri, Columbia, Missouri.

FIGURE 2. Cutting showing the same type of rot 48 hours after inoculation with Erwinia carotovora.

stems over the cut surface of potato slices. Within 10 hours the potato slices were badly decayed and displayed all the symptoms of soft rot caused by Erwinia carotovora. Dilution plates made from the rotted potato tissue yielded bacterial colonies apparently identical with those isolated directly from the poinsettias.

The bacteria isolated from the poinsettias have been partially characterized[3] with the following results:

1) Colonies formed on nutrient agar were gray-white, round, raised, smooth, glistening, and had entire margins,
2) Gram-negative staining reaction,
3) Motile (type of flagellation not determined),
4) Produced acid and gas from glucose, and
5) Grew both aerobically and anaerobically.

INOCULATION EXPERIMENTS

Nutrient broth cultures of both isolates and of a known isolate of Erwinia carotovora (A.T.C.C. 8061) were prepared, and freshly made poinsettia cuttings of the variety Barbara Ecke Supreme were inoculated by dipping the cut ends into the broth cultures. Control cuttings were dipped into sterile broth. All cuttings were incubated under moist chamber conditions at room temperatures (about 80° F) for 48 hours and then examined. The control cuttings were still perfectly healthy and firm, but all of the other cuttings were badly decayed. One of the cuttings inoculated with the known isolate of E. carotovora is shown in Figure 2. Reisolations from all three groups of decayed cuttings yielded bacteria which were apparently identical in colonial characteristics.

Some of the original cuttings were dried on a paper towel at the time received and held at room temperature and humidity conditions on a shelf in the lab. Six weeks later small portions of the dried decayed material were dropped into tubes of sterile nutrient broth. Within 48 hours the broth was cloudy and when used to inoculate other healthy cuttings it caused the typical rapid decay.

---

[3]The author is indebted to Dr. John E. Peterson, Assistant Professor of Botany, University of Missouri, for carrying out these tests.

Tests have also been completed to check the relative susceptibility of cuttings 1) made with the basal cut just below the nodal plate compared with 2) making the cut above the plate which results in a long hollow cylinder at the base of the cutting. No appreciable differences in susceptibility were noted. Cuttings made in either fashion decayed with equal rapidity.

DISCUSSION

The grower who submitted the cuttings felt that he had picked up the original inoculum from the rooting hormone powder used to treat the cuttings. However, repeated tests using samples of the powder applied directly to healthy cuttings have resulted in no decay. Likewise, after several attempts, no rot has resulted following inoculation of cuttings with broth cultures prepared by adding small quantities of the suspected hormone powder.

A conference with the grower suggested another possible source of the original inoculum. Special care had been taken to keep the cuttings clean during handling. The work table where trimming was done was covered with clean plastic. The propagation bench had just been built (using new lumber) and was being used for the first time. It had been filled with new perlite just before sticking the cuttings. However, the wet cuttings were being carried from the stock plant-producing area to the work table and from the work table to the propagation bench in old greenhouse flats containing only a sheet of clean newspaper in the bottom.

Since this newspaper soon became soaked, the flats could have been the source of trouble if they had contained any old plant material rotted by E. carotovora. This conjecture was borne out further by the fact that loss of additional cuttings was almost completely eliminated by treating all working areas with formaldehyde and substituting new, clean cardboard boxes for the old flats used for carrying the cuttings. E. carotovora is widely distributed and is a frequent inhabitant of decaying plant material. Our tests have shown that such pathogenic bacteria may remain infectious in dried plant tissue for at least 6 weeks -- how much longer we do not know.

Since a search of the literature indicates no previous record of Erwinia carotovora as a known pathogen of poinsettia, it is believed that this paper represents the first report of such an occurrence.

CONTROL

In view, particularly, of the great rapidity with which the pathogen operates, the only control that could be suggested at this time is the use of careful sanitation measures during propagation. This will prevent initial inoculation of the cuttings and will result in complete control of the disease.

MISSOURI AGRICULTURAL EXPERIMENT STATION, COLUMBIA

OCCURRENCE OF SOUTHERN CORN RUST IN WISCONSIN

M. S. Pavgi and A. L. Flangas

The two rusts predominant on corn are the common corn rust incited by Puccinia sorghi Schw. and the southern corn rust incited by Puccinia polysora Underw. A third rust incited by Angiopsora zeae Mains has been sparsely reported on corn only from tropical areas of Central and South America. The common rust is widely distributed in corn-growing regions throughout the world (1). In North America it has been an important disease of sweet and dent corn since the fungus was first identified and described by Schweinitz in 1832. The importance of southern rust on corn in North America, however, is of recent occurrence.

The association of corn and teosinte as hosts for Puccinia polysora was first reported by Cummins (3) in 1941 based on collections from South America. Subsequent surveys confirmed its occurrence in tropical areas of the western hemisphere including Central and South America, Mexico, and Puerto Rico (2, 3). Since then, however, its widespread occurrence has been reported. During the past decade it has been consistently reported from East and West African regions (4, 7, 9) as well as from the Philippine Islands (8), Malaya (5) and Thailand (11, 14) in South East Asia. The oldest American record of P. polysora parasitizing corn dates back to 1879 or perhaps earlier as evidenced by re-examination of a collection from Newton, Massachusetts. This rust has been occasionally collected from the southern States of Louisiana, South Carolina, Florida, Oklahoma, Mississippi, and Texas (14), but erroneously identified as P. sorghi. In 1949 Ullstrup (12) reported its occurrence on corn along the Ohio river in Indiana which was new for the North Central region. The presence of southern corn rust in North America prior to 1949 apparently is precluded, due to its association with P. sorghi, the incitant of common rust.

This year the southern rust has been observed in corn fields around Madison, Wisconsin. This is the first report of its occurrence in the State of Wisconsin. The wet climatic conditions prevalent during July and August in this area provided ideal environmental conditions for infection and spread of the rust pathogen, resulting in severe attack in several of the fields surveyed. Observations on corn in South Africa indicate that the disease is far less severe if the rainfall is scanty during July and August (4). Humidity seems to favor disease severity (10).

Field symptoms appear on leaves and leafsheaths of the plant before the tassels emerge (Fig. 1). The oval uredia are abundant, pustulate, buff brown in color, amphigenous, covered under the epidermis, and uniformly distributed. The uredospores are characteristically yellowish golden (in contents), oval to ovate and with hyaline, finely verrucose walls; with four to five almost equitorial germ pores, the spores measure 28-37μ x 20-26μ (Fig. 2)

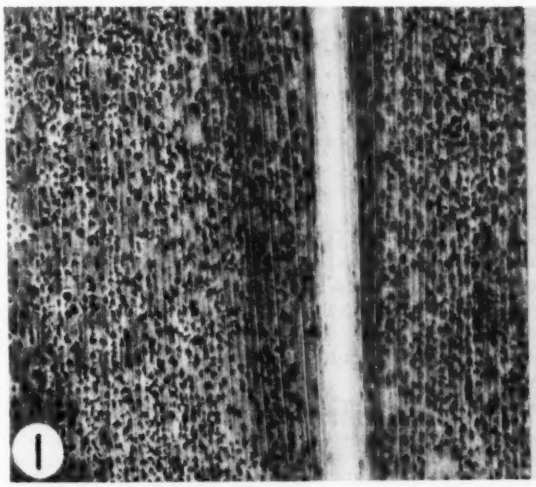


FIGURE 1. Portion of infected corn leaf showing abundance of indehiscent telia of Puccinia polysora on the undersurface.

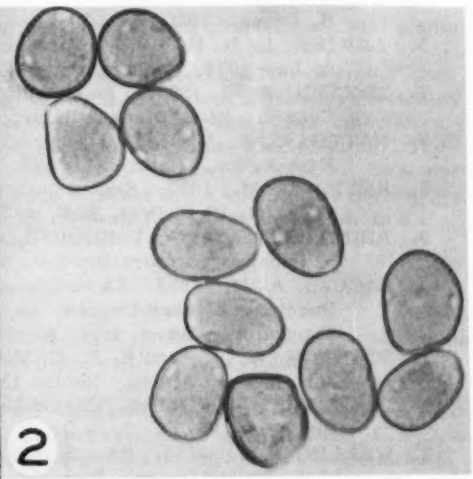


FIGURE 2. Uredospores showing germ pores.

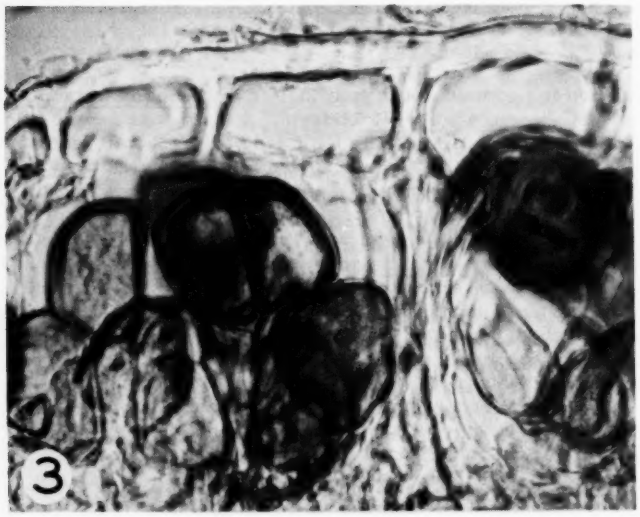


FIGURE 3. Freehand cross section through a telium.

Telia are invariably amphigenous, produced as minute, linear, bluish-black, confluent, irregular-shaped sori covered by the epidermis. The telium in cross section (Fig. 3) shows a raised indehiscent, subepidermal sorus containing chestnut brown, short pedicelled, densely clustered teliospores with little apical thickening of the wall. The apex is frequently irregular. Spores measure 31-39μ x 21-26μ. Mesospores are abundant, measuring 23-29μ x 15-23μ in range.

Both rusts have been considered of minor importance as diseases of corn. In a conservative estimate sampled from five north central States, Wallin (13) computed a loss of 20 million bushels of grain in 1950 from common rust alone. Although no data are available for the annual losses in grain yield, lately the rusts have often been assuming epiphytotic proportions (6, 13). Frequently the damage is enhanced as these rusts hasten drying of the leaves prematurely. Incidence of this rust reported here adds to the complex problem of disease control and represents a potential danger to the economic production of corn in North America.

Literature Cited

1. COMMONWEALTH MYCOL. INST. 1953. Distribution maps of Plant Diseases. No. 279. Puccinia sorghi Schw.
2. COMMONWEALTH MYCOL. INST. 1954. Distribution maps of Plant Diseases. No. 237. Puccinia polysora Underw. (2nd Ed.)
3. CUMMINS, G. B. 1941. Identity and distribution of three rusts of corns. Phytopathology 31: 856-857.
4. DEIGHTON, F. C. 1952. Plant Pathology Section Report. Dept. Agr. S. Leone. 1950. pp. 20-21.
5. LARTER, L. N. H. 1956. Notes on current investigations (research) April to June 1956. Malaya Agric. J. 39: 220-227.
6. McKEEN, W. E. 1951. An exceptionally early outbreak of corn rust in Canada. Plant Disease Reptr. 35: 367.
7. NATTRASS, R. M. 1953. Occurrence of Puccinia polysora Underw. in East Africa. Nature 171: 527.
8. REYES, G. M. 1957. Recent outbreaks of maize rust in the Philippines. F. A. O. Plant Prot. Bull. 6: 39-40.
9. RHIND, D., J. M. WATERHOUSE, and F. C. DEIGHTON. 1952. Occurrence of Puccinia polysora Underw. in West Africa. Nature 169: 631.
10. SACCAS, A. M. 1955. La rouille americaine du Mais (Zea mays L.) due a' Puccinia polysora Underw. au cameroun et en Afrique Equitoriale Francaise. Agron. trop. Nogent 10: 499-522.
11. STANTON, W. R., and R. H. CAMMACK. 1956. Studies on Puccinia polysora in West Africa. Robigo 1956. pp. 19-21.
12. ULLSTRUP, A. J. 1950. Corn diseases in Indiana in 1949. Plant Disease Reptr. 34: 98-99.
13. WALLIN, J. R. 1951. An epiphytotic of corn rust in the north central region of the United States. Plant Disease Reptr. 35: 207-211.
14. WOOD, J. I., and B. R. LIPSCOMB. 1956. Spread of Puccinia polysora with bibliography on the three rusts of Zea mays. Agricultural Research Service, United States Dept. of Agr., Special Publ. No. 9. 59 pp.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF WISCONSIN, MADISON, WISCONSIN

HELMINTHOSPORIUM ROSTRATUM DRECHS. ON RICE IN INDIA[1]

S. B. Chattopadhyay and C. Dasgupta[2]

Summary

Helminthosporium rostratum Drechs. was observed to incite small oval spots (2-3 x 1-1.5 mm) on leaves of rice in West Bengal, India. Isolations from infected leaves showed presence of two types of H. rostratum. Pathogenicity and morphology of both were studied. Both types were observed to be pathogenic when inoculated on leaves of 3-week-old seedlings of five varieties of rice. Study of morphology showed that both the types resembled one another in essential characters except macroscopic growth features and details of conidial shape and size.

INTRODUCTION

Helminthosporium rostratum Drechs. was first described on Eragrostis cilianensis (E. major) (3). Since then in the United States it has been reported on corn (4, 8, 9), Sorghum vulgare (9), Sorghum vulgare var. sudanense (9), Pennisetum glaucum (9, 5), Paspalum boscianum (4), Saccharum officinarum (6), and Cymbopogon citratus (6). The fungus has also been reported from the Gold Coast on corn (1, 2) and South Africa as inciting foot rot of wheat seedlings (7).

In the course of examining leaf spots caused by Helminthosporium oryzae, a type of spot differing from those caused by H. oryzae was observed. The spots were small, oval, delimited by veins, faint brown to straw colored and 2-3 x 1-1.5 mm in size. Isolations from these spots showed the presence of two distinct types of Helminthosporium sp. resembling one another in rostrate character of conidia. These were identified as Helminthosporium rostratum Drechs. and confirmed at the Commonwealth Mycological Institute, Kew, England.

H. rostratum was not previously reported on rice, nor from India. The morphology and pathogenicity of the fungus were investigated.

MATERIALS AND METHODS

Isolations were made from typical spots on rice leaves of varieties Patnai 23, Badkalamkati 65, Nagra 41/14, collected from different parts of West Bengal, India. The small spots from infected leaves were surface-sterilized and transferred to oatmeal agar slants and incubated at 26° C. Monoconidial cultures were made and the morphological characters demonstrated two distinct types designated as Isolate 1 and 2.

Morphology of the two types was studied on the host and on corn meal and oatmeal media in 10-cm Petri plates at temperatures of 21°, 26°, 31°, and 36° C.

Inoculations with the two types were studied on seedlings of five varieties of rice Patnai 23, Badkalamkati 65, Nagra 41/14, Dular and Kele. Leaves of the seedlings were inoculated in three-leaf stage with a conidial suspension of the two types. The plants after inoculation were covered with bell jars for 24 hours to maintain the humidity during the infection. The pathogenicity tests were made in August when the disease was prevalent in nature. Data were taken on percentage of plants infected, average number of spots in 1-cm length of leaves, size and color of spots. Reisolations were made from the representative leaf spots and, in all cases, the isolate used in inoculation was recovered.

OBSERVATIONS

Data on the pathogenicity test are presented in Table 1. Both isolates were pathogenic to five varieties of rice. Isolate 1 appeared somewhat more virulent than Isolate 2.

[1] Authors gratefully acknowledge the help rendered by Professor J. G. Dickson, Professor of Plant Pathology, University of Wisconsin and Agent, Field Crops Research Branch, A. R. S., U. S. D. A. in the preparation of this paper for publication. They wish to thank the Director, Commonwealth Mycological Institute, Kew, England for confirmation of the identification of the isolates.

[2] Mycologist and Research Assistant, Mycology Section, State Agricultural Research Institute, Calcutta, India, respectively.

Table 1. Data on infection and symptom incited by two isolates of H. rostratum on seedlings of five varieties of rice.

Variety	Isolate	Percentage of plants infected	Average number of spots in 1-cm length of 50 leaves	Size of spots (in mm)	Color
Patnai 23	1	67	112	1-2.5 x 0.8	Faint brown
	2	59	98	1-2.5 x 0.8-1	Faint brown
Badkalamkati 65	1	77	128	1-3 x 0.8-1	Faint brown
	2	66	100	1-2.5 x 0.8-1	Faint brown
Nagra 41/14	1	67	112	1-2.5 x 0.5-0.8	Faint brown
	2	48	80	1-2.5 x 0.5-0.8	Faint brown
Dular	1	55	98	1-2 x 0.5	Faint brown
	2	55	100	1-2 x 0.5	Faint brown
Kele	1	48	88	1-2.5 x 0.5-0.8	Faint brown
	2	41	64	1-2 x 0.5-0.8	Faint brown

Table 2. Data on size and septation of conidia produced by two isolates of H. rostratum on host and artificial media.

Isolate	Media	Size of conidia (in μ)	Average size of conidia (in μ)	Septation	Mode of septation
1	Host	9.6- 99.6 x 7.7-15.3	47.5 x 12.6	0-11	5-7
	Oatmeal agar	17.2-149.4 x 7.7-15.3	55.9 x 12	1-12	5-8
	Corn meal agar	11.5-124.5 x 7.7-15.3	54.0 x 11.1	0-11	5-8
2	Host	11.5-120.6 x 7.7-15.3	66.8 x 10.9	0-11	6-8
	Oatmeal agar	17.5- 99.6 x 9.6-15.3	50.6 x 14.1	0-10	5-7
	Corn meal agar	17.2- 77.6 x 9.6-15.3	53.4 x 14.6	1-11	4-6

Table 3. Diameter of colony (in mm) of the two isolates of H. rostratum after 72 and 168 hours of inoculation on oatmeal and corn meal agar at different temperatures.

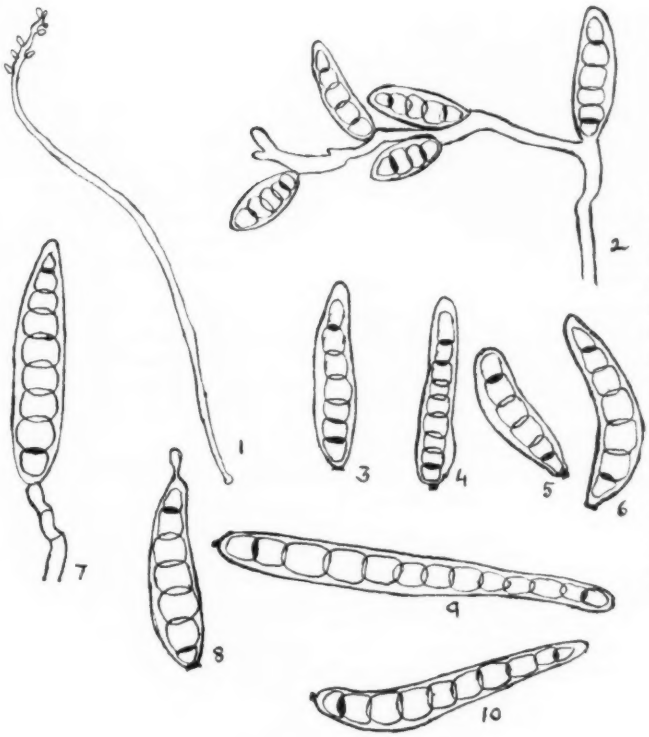
Media	Isolate	Temperatures							
		21° C		26° C		31° C		36° C	
		72 hours	168 hours	72 hours	168 hours	72 hours	168 hours	72 hours	168 hours
Oatmeal agar	1	44	93	41	99	37	88	36	87
	2	39	88	38	97	38	91	35	85
Corn meal agar	1	40	89	38	96	43	85	34	85
	2	39	81	36	94	41	92	32	83

Morphology of the Isolates

Conidiophores: In both isolates, conidiophores on the host emerge through the stomata or between epidermal cells singly or in groups of 2 to 4. They are dark olivaceous, closely septate near the apex, with a swollen basal cell, and bear conidia in succession near the apex. Conidiophores of Isolate 1 on the host are long, trailing and measure 262.8-1044μ x 5.8-9.6μ, averaging 482.5 x 6.8μ(Figs. 1, 2). Those on artificial media are straight, much shorter, and measure up to 400μ in length (Fig. 7).

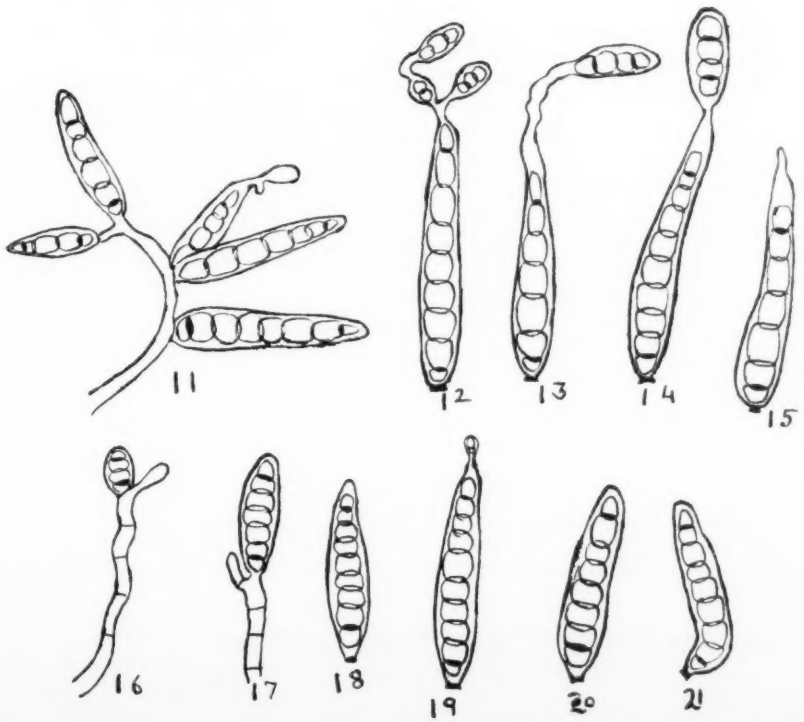
Conidiophores of Isolate 2 on the host (Fig. 11) and artificial media (Fig. 16) are straight with tendency of branching, knotted or geniculate near apex and measure up to 570μ in length on the host, averaging 312 x 6.5μ, while those produced on the artificial media are up to 300μ in length.

Conidia: Conidia of both the isolates (Figs. 3-10, 12-21) produced on the host and on artificial media are dark olivaceous, straight, oblanceolate to frequently curved, widest near the base to the middle, usually tapering from base to apex which is blunt and rounded. The basal cell shows distinct protruding hilum, the basal and distal septa are thicker and more prominent.



FIGURES 1-10. Conidiophore and conidia of Isolate 1 of H. rostratum. 1. Trailing conidiophore on the host. 2. Conidiophore with conidia on the host. 3-6. Conidia formed on the host. 7-10. Conidia formed on oatmeal agar.

FIGURES 11-21. Conidiophore and conidia of Isolate 2 of H. rostratum. 11. Conidiophore with conidia on the host. 12-15. Conidia formed on the host. 16-21. Conidia formed on oatmeal agar.



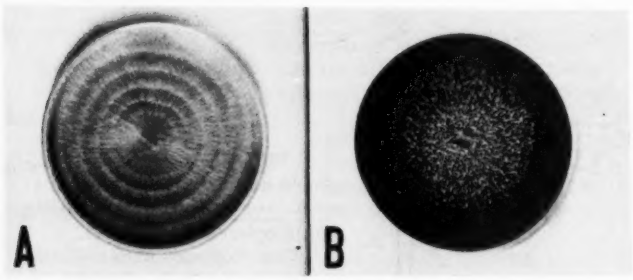


FIGURE 22. Macroscopic growth features of the two isolates of H. rostratum, on oatmeal agar at 26° C. A--Isolate 1. B--Isolate 2.

Conidia of Isolate 2, on the host particularly, are strongly rostrate in the apical end and show tendencies to proliferate even when attached to conidiophore (Figs. 12-15). Conidia of Isolate 1 are feebly rostrate on artificial media only (Fig. 8). Measurements and septation of conidia of the two isolates produced on the host and on two artificial media are given in Table 2.

Growth on Artificial Media: Data on the diameter of colony after 72 and 168 hours of inoculation on oatmeal agar and corn meal agar in Petri plates at 21°, 26°, 31°, and 36° C are given in Table 3. There is no difference in rate of growth between the two isolates either in the two media or at different temperatures.

Macroscopic Growth Features: At 26° C on both media both isolates showed good aerial mycelial growth which was appressed to granular in texture and smoky gray to dark gray in color. Zonation was very prominent in Isolate 1 (Fig. 22A) while it was practically absent in Isolate 2 (Fig. 22B). Mycelial development and coloration were more intense at 21°. At 31° and 36° mycelial growth was sparse and light gray in color.

DISCUSSION

It is evident that both types of H. rostratum are pathogenic to rice leaves, in which they incite small oval to linear light colored spots. Both types resemble each other closely except in zonation of mycelial growth and detailed character of conidia. The Isolate 1 type may be regarded as a morphological variant of typical H. rostratum.

Literature Cited

1. BUNTING, R. H. 1927. Local cereal diseases in the records of the mycological division. Yearbook Gold Coast Dept. of Agr. Bull. 7: 25-27.
2. BUNTING, R. H. 1928. Black auricle disease of maize: Fungi affecting graminaceous plants of the Gold Coast. Gold Coast Dept. of Agr. Bull. 10: 19-20.
3. DRECHSLER, C. 1923. Some graminicolus species of Helminthosporium. J. Agr. Research 24: 641-740.
4. LEFEBVRE, C. L., and H. W. JOHNSON. 1941. Collections of fungi, bacteria and nematodes of grasses. Plant Disease Reptr. 25: 556-579.
5. LUTTRELL, E. S. 1954. Diseases of pearl millet in Georgia. Plant Disease Reptr. 38: 507-514.
6. PARRIS, G. K. 1950. The Helminthosporia that attack sugar cane. Phytopathology 40: 90-103.
7. PUTTERILL, K. M. 1954. Some graminicolus species of Helminthosporium and Curvularia occurring in South Africa. Bothalia 6: 347-378.
8. ULLSTRUP, A. J. 1954. Helminthosporium diseases on corn. Plant Disease Reptr. Suppl. 228: 118-119.
9. YOUNG, G. Y., C. L. LEFEBVRE, and A. G. JOHNSON. 1947. Helminthosporium rostratum on corn, sorghum and pearl millet. Phytopathology 37: 180-183.

STATE AGRICULTURAL RESEARCH INSTITUTE, CALCUTTA, INDIA

AN OUTBREAK OF PHLOEM NECROSIS IN ALABAMA

E. A. Curl, L. L. Hyche and N. L. Marshall[1]

Elm phloem necrosis, a virus disease transmitted by the leaf hopper Scaphoideus luteolus, is widely distributed over approximately 16 States including Alabama. The disease has not yet caused such general concern or great damage in the South as is common in some of the mid-western States, although an outbreak of considerable proportion was observed at Lindale and Rome, Georgia (3) in 1951. This outbreak represented a probable southward spread of about 50 miles from a previously known occurrence near Chattanooga, Tennessee. No cases of phloem necrosis were found by Bretz and Swingle (1) in 1946 during an exploratory survey in Georgia and Alabama. However, in 1949 Swingle et al. (4) included these two States in a map showing distribution of the disease. Through personal correspondence it was learned that R. R. Whitten and Charles Griswold identified a case of phloem necrosis in Chambers County, Alabama near the Georgia State line in 1948. A disease distribution map published by Carter (2) in 1950 includes this county in Alabama along with other limited infested areas in Georgia and Mississippi.

Early in July 1959 officials[2] of Maxwell Air Force Base, Montgomery, Alabama became alarmed at a sudden and rapid dying of elms on Base property. Subsequently the area was examined and certain observations made. It was estimated that at least 40 trees of an estimated 500 elms (Ulmus alata Michx.) on the Base were dead or dying. Most of the trees died so rapidly that the leaves turned brown and remained attached. A few of the trees were partially defoliated (Fig. 1). In one small area of approximately one-half acre, 14 of 20 elms were

FIGURE 1. One of approximately 40 elm trees, Ulmus alata Michx., infected with phloem necrosis on the Maxwell Air Force Base, Montgomery, Alabama.

either dead or in various definite stages of decline. Most of these trees had been planted in rows, and the disease apparently progressed from one end of a row to the other, indicating root-graft transmission.

The wood of branches taken from diseased trees failed to reveal any of the brown streaking characteristic of Dutch elm disease, and isolation attempts were negative. The European elm bark beetle, Scolytus multistriatus (Marsh.)[3], and its typical galleries were found in large numbers in infected trees. However, the Dutch elm disease fungus, Ceratocystis ulmi (Buis.)

---

[1] Associate Plant Pathologist, Assistant Entomologist and Assistant Professor of Plant Pathology, respectively.

[2] The authors are grateful to Lt. Colonel M. J. Beeghly, Installations Engineer, and other officials of the Maxwell Air Force Base for calling attention to the disease and for close cooperation during the observations that were made.

[3] The elm bark beetle was identified by W. H. Anderson, Insect Identification and Parasite Introduction Research Branch, Plant Industry Station, Beltsville, Maryland.

Moreau, was not found in these galleries. Further examination revealed the butterscotch color characteristic of phloem necrosis in the inner bark of several trees; also, the odor of wintergreen was evident in these trees. Therefore, the disease was diagnosed as phloem necrosis, a diagnosis which later was verified by A. F. Verrall, Chief, Division of Forest Disease Research , Southern Forest Experiment Station, New Orleans, Louisiana.

At the present time the disease seems to be confined to the Air Force Base, although there are many elm trees in the city of Montgomery. This infestation lies approximately 80 miles southwest of the earlier case in Chambers County and about 250 miles from an infested area in Jackson, Mississippi.

Officials of the Maxwell Base are planning control measures that will consist of removal of all dead and dying elms on the base and either an annual spray program or replacement of killed elms with varieties resistant to phloem necrosis.

Literature Cited

1. BRETZ, T. W., and R. U. SWINGLE. 1946. Known distribution of phloem necrosis of the American elm. Plant Disease Reptr. 30: 156-159.
2. CARTER, J. C. 1950. Status of oak wilt and elm phloem necrosis in the midwest. Arborists' News, 15: 45-51.
3. MILLER, J. H., W. A. CAMPBELL, and G. E. THOMPSON. 1954. Diseases and insects affecting the commonly planted trees and shrubs in Georgia. Plant Disease Reptr. 38: 362-369.
4. SWINGLE, R. U., R. R. WHITTEN, and H. C. YOUNG. 1949. The identification and control of elm phloem necrosis and Dutch elm disease. Ohio Agr. Exp. Sta. Spec. Circ. 80.

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY AND DEPARTMENT OF ZOOLOGY-ENTOMOLOGY, ALABAMA POLYTECHNIC INSTITUTE, AUBURN, ALABAMA

AN IMPROVED METHOD OF TESTING THE TOLERANCE OF STRAWBERRY VARIETIES AND NEW SELECTED SEEDLINGS TO VIRUS INFECTION[1]

P. W. Miller[2]

It is important for plant breeders to know the relative tolerance of a promising new strawberry selection to virus infection before it is released for general distribution. In the past, the prevailing method of testing its tolerance has been to plant the selection in the field in the midst of one or more infected susceptible varieties and to depend upon viruliferous insects to infect it. Later the selection is examined and if virus symptoms and degeneration are visible it is presumed susceptible; if not, it is considered to possess a degree of tolerance to the virus, or virus complex, involved.

There are several objections to this method of determining virus tolerance. The virus or virus complex present may not be definitely known or there may not have been any viruliferous insects present to transmit the virus or virus complex. Then too, some viruses, such as the latent A virus, are not transmitted by aphids or other known insects.

A more positive and quicker method of evaluating a selection for virus tolerance is needed. A discussion of methods recently devised to test varieties and selected seedlings and the results of some typical inoculations follows.

METHODS AND MATERIALS

The plants tested were grown in 6- to 8-inch pots in a fertile soil in a greenhouse until four or more mature or semi-mature leaves were present. Three plants of each variety or selection were then inoculated with known virus components, using Miller's modification of Bringhurst and Voth's excised leaf-petiole graft technique[3]. The virus components were introduced singly and/or in combination of two or more. Two leaf grafts of each component were made in each plant. The inoculated plants were then placed in a polyethylene film moist chamber for about 10 days, after which they were incubated in the greenhouse at an average temperature of 70° F. Most of the inoculated plants were later indexed to determine whether all components were transferred.

Virus symptoms ranging from a slight shortening of the petioles and a slight to marked leaf dwarfing, leaf curling, chlorosis, and necrosis generally appeared in 3 to 9 weeks. Uninoculated plants of the same variety or selected seedling were maintained as checks for comparison. The virus "readings" were generally made first under greenhouse conditions. Later, the inoculated plants were transplanted to the field to determine the rate of degeneration there.

RESULTS

The results of some typical tests using the procedures described are given in Tables 1 and 2.

The reactions of three inoculated plants each of the Marshall variety and certain selected seedlings to individual virus components are compared in Table 1 while in Table 2 are given the comparative reactions of the Marshall and the selected seedlings inoculated with certain virus complexes.

It is evident from the data given in these tables that virus symptoms usually appear relatively early after grafting when the techniques outlined are used.

It would appear from the results that virus tolerance is largely relative and, in most cases, it can be "broken down" if a sufficient number of the different types of components are introduced into the plant (Fig. 1). Thus, U. S.-Oreg. selection 2433 is apparently tolerant to all of the single viruses tested (latent A, latent C, mottle, crinkle, veinbanding and mild yellow edge), but moderate to severe symptoms appeared when plants of this selection were inoculated with latent A + mild yellow edge + veinbanding + crinkle virus components.

---

[1]Cooperative investigations by Crops Research Division, Agricultural Research Service, United States Department of Agriculture and Oregon Agricultural Experiment Station, Corvallis, Oregon.

[2]Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

[3]Miller, P. W. 1958. Comparative efficiency of excised leaf-petiole grafts and stolon grafts for transmitting certain strawberry viruses. Plant Disease Reptr. 42: 1043-1047.

Table 1. Comparative susceptibility of some selected strawberry seedlings and virus-free plants of Marshall inoculated with single viruses by excised leaf-petiole grafting, 1958-59.

Virus component and variety or selection inoculated	Results of reindexing[a]	Relative severity of symptoms[b]	Incubation period (days)
Latent A			
U. S.-Ore. 2414	+	None	--
U. S.-Ore. 2433	+	None	--
Marshall	+	None	--
Latent C			
U. S.-Ore. 2414	+	None	--
U. S.-Ore. 2433	+	None	--
Marshall	+	None	--
Mottle			
U. S.-Ore. 2414	+	None	--
U. S.-Ore. 2433	+	None	--
Marshall	+	None	--
Crinkle			
U. S.-Ore. 2414	+	None	--
U. S.-Ore. 2433	+	None	--
Marshall	+	Moderate	77
Veinbanding			
U. S.-Ore. 2414	+	None	--
U. S.-Ore. 2433	+	None	--
Mild yellow edge			
U. S.-Ore. 2414	+	None	--
U. S.-Ore. 2433	+	None	--
Marshall	+	Mild	--

[a] + = Virus introduced "recovered" on reindexing to proper indicators.
[b] Condition 120 days after grafts made; "readings" made in greenhouse before plants transferred to field.

FIGURE 1. U. S.-Oreg. selection 2531 infected with the latent A + veinbanding + mottle + crinkle + mild yellow-edge viruses by excised leaf-petiole grafts; 30 days after grafts made.

Table 2. Comparative susceptibility of some selected strawberry seedlings and virus-free plants of Marshall inoculated with certain virus complexes by excised leaf-petiole grafts, 1958-59.

Virus complex and variety or selection inoculated	Results of reindexing[a]	Relative severity of symptoms[b]	Incubation period (days)
Latent A + mottle			
U. S.-Ore. 2414	+	None	--
Latent A + veinbanding			
U. S.-Ore. 2414	+	None	--
Latent A + crinkle			
U. S.-Ore. 2414	+	None	--
Latent A + mild yellow edge			
U. S.-Ore. 2416	+	None	--
Latent A + mild yellow edge + veinbanding			
Marshall	--	Moderate	28
Latent A + crinkle + mild yellow edge			
Marshall	--	Severe	45
Latent A + veinbanding + mottle + mild yellow edge			
U. S.-Ore. 2234	--	Moderate	--
U. S.-Ore. 2331	--	Moderate	--
U. S.-Ore. 2414	--	Severe	--
Latent A + mild yellow edge + veinbanding + crinkle			
U. S.-Ore. 2433	--	Severe	74
Latent A + mottle + veinbanding + mild yellow edge + crinkle			
U. S.-Ore. 2326	--	Severe	72
U. S.-Ore. 2426	+	None	--
U. S.-Ore. 2433	--	Moderate	13
U. S.-Ore. 2531	--	Moderate	20
Latent A + latent C + mottle + veinbanding + mild yellow edge			
U. S.-Ore. 2416	--	Moderate	40
Latent A + latent C + mottle + veinbanding + crinkle + mild yellow edge			
U. S.-Ore. 2247	--	Severe	72
U. S.-Ore. 2434	--	Severe	55

[a] + Virus complex introduced "recovered" on reindexing to proper indicators.
[b] Condition 120 days after grafts made; "readings" made in greenhouse before plants transferred to field.

It is noteworthy that one of the selections, U. S.-Oreg. 2426, did not develop any noticeable virus symptoms even when inoculated with the whole "spectrum" of virus components tested. This selection is not immune, however, as inoculated plants of this selection were reindexed and found to have been successfully infected.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE AND OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS

EFFECT OF STORAGE TIME AND TEMPERATURE ON CONTROL OF SEED-BORNE SAFFLOWER RUST BY VOLATILE MERCURY FUNGICIDES AND THE EFFICACY OF CERTAIN SEED PROTECTANTS AND ANTIBIOTICS

C. A. Thomas

Summary

Storage time and temperature had small and consistent effects on control by the volatile mercury fungicides tested. The mercury materials gave as good control as the seed protectants or better at comparable dosages and gave perfect control in certain storage tests.

INTRODUCTION

Safflower, Carthamus tinctorius, is a relatively new oilseed crop adapted to the small-grain areas of the western United States. Rust, Puccinia carthami Cda., is usually serious and prevalent in plantings on surface-irrigated land. The fact that this autoecious, brachyform rust is readily transmitted by teliospores borne on the surface of the seed probably accounts for its widespread distribution (2, 5).

The lack of suitable resistant varieties of safflower (6) or other effective and feasible control measures has focused attention on the use of rust-free seed and crop rotation as exclusionary methods of control. Commercial seed treatment with volatile mercury compounds has not been completely effective in controlling seed-borne teliospores. Complete control of seed-borne teliospores is desirable because it would eliminate a primary source of inoculum for leaf infection. This paper reports results of tests conducted in the greenhouse at Beltsville, Maryland since 1956 to determine the efficacies of seed treatment materials as well as to study the effects of storage duration and temperature on control by volatile mercuries.

MATERIALS AND METHODS

Safflower seed of the variety Pacific 2 was used in all tests. Teliospores were collected each summer from naturally infected plants in Arizona or California and were stored in vials at 15° C. Seed in lots of 500-cc was heavily infested by shaking with 2 grams of teliospores.

The seed treatment materials studied were Orthocide 75 (75 percent captan, N-(trichloromethylthio)-4 cyclohexene-1, 2-dicarboximide), Arasan 75 (75 percent thiram, bis(dimethylthiocarbamoyl) disulfide), Spergon (96 percent chloranil, tetrachloro-p-benzoquinone), Phygon (50 percent dichlone, 2, 3-dichloro-1, 4-napthoquinone), Manzate (70 percent maneb, manganous ethylene bis-(dithiocarbamate), Dithane Z-78 (65 percent zineb, zinc ethylene bis-(dithiocarbamate), PCNB (20 percent pentachloronitrobenzene), Ceresan M (7.7 percent N-(ethylmercuri)-p-toluenesulfonanilide), Ceresan 75 (2.8 percent ethylmercuri 2, 3-dithydroxypropyl mercaptide and 0.6 percent ethylmercuri acetate), and Panogen 15 (2.2 percent cyano (methylmercuri) guanidine. Three antibiotics, oligomycin, Acti-dione, and a complex known as F-17, also were used as seed treatments.

Artificially infested seeds in lots of 500-cc were treated with the materials listed. Ceresan M was applied as a slurry; Ceresan 75 and Panogen 15 were applied as liquids. Seeds were soaked for 1 hour in solutions containing the antibiotics and then dried at room temperature. The other materials were applied as dusts.

In some tests seeds treated with the volatile mercury materials were stored in sealed flasks prior to planting for 1 day to 6 months. Storage temperatures of 5°, 10°, 15°, 20°, 25°, 30°, and 40° C were used in conjunction with certain dosage rates and storage periods. In tests in which the mercury compounds were compared with the seed protectants and antibiotics storage was at 25° for 24 hours. Seeds treated with other materials were planted immediately after treatment. Most materials were tested at dosages of 1/2 to 4 ounces/bushel of seed. Certain chemicals were tested at maximum dosage to determine phytotoxicity and maximum control possible.

Most tests were run two or more times and included seven replications of 200 seeds each for each treatment. Treated seeds were planted in moist steamed soil in flats held at 5° C for 1 week in a cold room and then at 20° for 4 weeks in a greenhouse. The rust-infected seedlings were counted.

Table 1. Control of Puccinia carthami on safflower seed by treatment with protectants, volatile mercuries, and antibiotics.

Treatment	Dosage (ounces/bushel)	Mean percentage of seedlings infected[a]
None	--	100.0
Arasan 75	1/2	24.8
Arasan 75	1	3.2
Arasan 75	2	2.0
Arasan 75	max.	1.8
Spergon	1/2	66.8
Spergon	1	28.0
Spergon	max.	3.0
Orthocide 75	1	30.0
Orthocide 75	max.	4.0
PCNB	max.	2.0
Phygon	1/2	15.2
Phygon	2	2.0
Phygon	4	0.62
Phygon	max.	0.25
Manzate	2	3.0
Dithane Z-78	max.	0.5
Ceresan M	1/2	5.0
Ceresan M	1	0.4
Ceresan M	4	0.2
Ceresan 75	1/2	8.7
Ceresan 75	1	1.1
Ceresan 75	4	0.4
Panogen 15	1/2	4.2
Panogen 15	1	1.2
Panogen 15	2	0.5
Panogen 15	3	0.2
Panogen 15	4	0.06

Treatment	Dosage (ppm)	Mean percentage of seedlings infected[a]
Oligomycin	500	22.6
	1000	19.1
	5000	9.5
Acti-dione	500	0.04
F-17	16000	10.0

[a]Average of two or more tests each consisting of seven replications of 200 seeds.

Table 2. Mean percentage infected seedlings[a] produced by infested seed treated with Panogen 15 at three dosage rates and held for 3 days in sealed storage at seven temperatures.

Dosage (ounces/bushel)	Storage temperature (°C)						
	5°	10°	15°	20°	25°	30°	40°
1	-	-	2.5	-	-	1.2	-
2	1.4	1.2	1.1	0.35	0.30	0.16	0.11
4	-	-	0.55	-	-	0.05	-

[a]Average of two or more tests each consisting of seven replications of 200 seeds.

RESULTS

Results of tests in which the efficacies of seed protectants, volatile mercuries, and antibiotics were compared showed that none gave perfect control (Table 1). All appeared to give good control at the higher dosages. Arasan 75, Spergon, and Orthocide 75 were less effective than other materials tested at the lowest dosage rates. Acti-dione, which reduced stand by 60 percent, was the only material that appeared phytotoxic.

Storage period and temperature had small and consistent effects on the number of rust-infected seedlings produced by seed treated with volatile mercury compounds. Test results showed that increasing the dosage of Panogen 15 and increasing the storage temperature resulted in reductions of the percentage of infected seedlings (Table 2). Seed treated with Panogen 15 at 2 ounces per bushel and stored at 25° C for 0, 1, 3, 7, and 30 days produced, respectively, 0.8, 0.5, 0.3, 0.1, and 0.07 percent infected seedlings. No infected seedlings were found when storage was for 2, 3, 4, or 6 months, although seedlings produced by untreated seed subjected to the same storage conditions were 100 percent infected. Similar results were obtained with Ceresan 75 and Ceresan M. No significant reduction in stand or other evidence of phytotoxicity was observed in any of the treatments.

DISCUSSION

Perfect control of teliospores on naturally infested seed might be easier to obtain than these tests indicate since the inoculum level probably would be considerably lower. Such a determination would be difficult to make in view of the large number of seedlings necessary when infection is less than 1 percent.

Wilson and Ark (7) reported perfect control with Acti-dione at a concentration as low as 1 ppm. They used seed from a lot that produced 8 percent infected seedlings. Acti-dione in the more extensive tests reported here, and under more severe conditions, gave good but not perfect control.

Storage period, storage temperature, and dosage rate appeared to be of particular importance for complete control of seed-borne teliospores by treatment with the volatile mercury materials. The effect of temperature was not unexpected. In 1943, Lehman (3) reported that ethyl mercury phosphate gave markedly better control of cotton seed molds at 28° than at 5° C. Arny and Leben (1), using a temperature of 20° to 25°, reported effective control of a seedling blight of oats with vapors of Ceresan M and Panogen. Purdy and Holton (4), however, found volatile products of Ceresan M ineffective for control of wheat bunt in tests at 15°. Standardization of treatment conditions in experimental tests with volatile mercury materials may be desirable.

Literature Cited

1. ARNY, D. C., and CURT LEBEN. 1954. Vapor action of certain mercury seed treatment materials. Phytopathology 44: 380-383.
2. CALVERT, O. H., and C. A. THOMAS. 1954. Some factors affecting seed transmission of safflower rust. Phytopathology 44: 609.
3. LEHMAN, S. G. 1943. Vapor action of certain fungicidal materials prepared for dusting cotton seed. Phytopathology 33: 431-448.
4. PURDY, LAURENCE H., and C. S. HOLTON. 1956. Vapor action of fungicides used in the control of wheat bunt. Phytopathology 46: 385-387.
5. THOMAS, C. A. 1952. Transmission of safflower rust on treated seed. Phytopathology 42: 108-109.
6. THOMAS, C. A. 1958. Reaction of safflower varieties to regional collections of Puccinia carthami. Plant Disease Reptr. 42: 1089-1090.
7. WILSON, EUGENE M., and PETER A. ARK. 1958. Acti-dione as a control of seed-borne safflower rust. Phytopathology 48: 640.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

RELATIVE RESISTANCE OF SOME STRAWBERRY VARIETIES, SPECIES, SELECTIONS AND SEEDLING POPULATIONS TO POWDERY MILDEW AT AGASSIZ AND ABBOTSFORD, BRITISH COLUMBIA

Hugh A. Daubeny[1]

Following the relatively cool and humid summer of 1959, powdery mildew, caused by Sphaerotheca macularis (Fr.) Magn., has been serious on strawberries throughout the Lower Fraser Valley area of British Columbia. In plantings at the Experimental Farm, Agassiz and the Small Fruit Substation, Abbotsford, 40 miles southwest of Agassiz, there has been a severe epidemic of the disease and it has been possible to obtain information on the relative resistance of varieties, species, selections and seedling populations to the fungus.

The five powdery mildew resistance ratings used were as follows: 10 = no mildew; 9 = trace of mildew but no leaf rolling; 8 = small amount of mildew with slight leaf rolling; 7 = leaves considerably mottled with some leaf rolling (this rating was a modification of that described by Miller and Waldo[2] as considerable mottle with little or no leaf rolling); 6 = much leaf rolling and some injury to lower surface (reddening and beginning necrosis); 5 = much leaf rolling accompanied by severe necrosis (injury severe enough to cause loss of leaves). With the exception noted, these were similar to the rating used by Miller and Waldo[2] and Orchard and van Adrichem[3].

Table 1 gives the powdery mildew resistance ratings on two different dates for varieties, species and selections growing at Abbotsford. Siletz, two selections of F. chiloensis (from Vancouver Island and from Oregon, respectively), F. ovalis (from Vancouver Island), and Agassiz seedlings 90 to 92 showed only a trace of mildew and thus can be classed as highly resistant to the disease.

Powdery mildew resistant ratings were taken September 28 for over 100 varieties or selections growing in the field at Agassiz. Siletz and a selection from the cross of Temple x Or. 2172 showed no mildew; Or. 2467 and Or. 2351 and eight Agassiz selections showed a trace of the disease. The high resistance rating for Or. 2467 is questioned since, at Abbotsford, its rating dropped from 8 on September 8 to 5 on September 29.

The seedling populations examined for powdery mildew resistance had been planted in red stele infested soil in greenhouse benches at Agassiz. Soon after the planting was completed in the latter part of August, a severe epidemic of mildew was noted. Also noted was the fact that the British Sovereign plants, which were set at regular intervals throughout the planting, were uniformly, though not severely, infected with the disease. These circumstances appeared to offer an ideal opportunity to screen the seedlings for possible resistance or immunity. Each seedling was examined and rated for severity of the disease. Subsequently, each seedling rated 10 was reexamined at five different dates within a 30-day period and, if the disease was found, given a lower rating. Table 2 gives the crosses and for each cross the number of seedlings examined, the average relative rating for powdery mildew resistance, and the number and percentage of seedlings rated 10 or free from the disease after the fifth examination.

As shown in Table 2, the cross of Siletz x Puget Beauty and its reciprocal gave relatively large numbers of seedlings which remained free from powdery mildew; also, average resistance ratings were higher for seedlings of these crosses than for those of any of the others. It is noteworthy that the crosses involving British Sovereign and either one of the two selections of F. chiloensis or the selection of F. ovalis gave few seedlings which remained free from powdery mildew. In the field, all three selections were similar to Siletz in their reaction to the disease and British Sovereign was similar to Puget Beauty. It may be that the genes for resistance in the selections from the species are in a relatively heterozygous state, whereas those in Siletz, which is derived in part from an Oregon selection of F. chiloensis, are in a more homozygous state. Also, the genes conferring some degree of resistance in Puget Beauty may be relatively more homozygous than those conferring a similar degree in British Sovereign. It is to be expected that parents more homozygous for resistance would, when crossed, produce larger numbers of resistant seedlings. From a practical viewpoint, seedlings selected for resistance to powdery mildew from the cross of Siletz x Puget Beauty or its reciprocal will be very useful in the future breeding program.

---

[1]Research Branch, Canada Department of Agriculture, Experimental Farm, Agassiz, British Columbia.

[2]Miller, P. W., and G. F. Waldo. 1957. Relative resistance of some strawberry varieties and selections to powdery mildew at Corvallis, Oregon. Plant Disease Reptr. 41: 23-24.

[3]Orchard, W. R., and M. C. J. van Adrichem. 1957. Relative resistance of some strawberry species, varieties, and selections to powdery mildew at Saanichton, British Columbia. Plant Disease Reptr. 41: 945-947.

Table 1. Relative resistance of some strawberry varieties, species, and selections to powdery mildew, Abbotsford Small Fruit Sub-Station, British Columbia, 1959.

Variety, species or selection	Resistance rating : Sept. 8	Sept. 29
Agassiz 90 (Agassiz x Brit. Sov.)	9	8
Ag. 92(Brit. Sov. $S._3$)	9	8
Fragaria chiloensis (Oregon)	9	8
F. chiloensis (Vancouver Island)	9	8
F. ovalis	9	8
Siletz	9	8
Ag. 89 (Agassiz x Brit. Sov.)	9	7
Ag. 93 (Brit. Sov. $S._3$)	8	8
Ag. 101 (Temple $S._2$)	8	8
Erie	8	8
Huxley	8	8
New York 386	8	8
Oregon 2234	8	8
Or. 2351	8	8
Saanich 4607-5	8	8
Senator Dunlap	8	8
Sparta Everbearing	8	8
Aberdeen	8	7
Ag. 44 (Brit. Sov. x Massey)	8	7
Alaska	8	7
Arrowhead	8	7
British Sovereign	8	7
Dorset	8	7
Essex	7	8
MacKenzie	8	7
N.Y. 219	7	8
N.Y. 418	8	7
Orland	8	7
Or. 2172	8	7
Or. 2247	8	7
Or. 2441	8	7
Puget Beauty	8	7
Red Coat	7	8
S-4607-9	7	8
Sparkle	8	7
Indra	7	
Ag. 100 (Temple S. x Brit. $Sov.)_2$	8	6
Dutch Sovereign	8	6
Or. 2451	8	6
Or. 2467	8	5
Ag. 39 (Brit. Sov. x Or. 1639)	7	7
Ag. 59 (Marshall x Pathfinder)	7	7
Ag. 87 ((Brit. Sov. x Or. 1639)S.)	7	7
Ag. 91 (Agassiz x Brit. Sov.)	7	7
Ag. 94 (Or. 1639 $S._2$)	7	7
Ag. 102 (Temple $S._2$)	7	7
Auchincruive	7	7
Brilliant Everbearing	7	7
Cambridge Early	7	7
Cambridge Vigour	7	7
Earlidawn	7	7
Empire	7	7
Jersey Belle	7	7
Maine 55	7	7

(continued)

Variety, species or selection	Resistance rating : Sept. 8	Sept. 29
Maryland 683	7	7
Monmouth	7	7
N.Y. 27	7	7
N.Y. 423	7	7
Or. 2175	7	7
Or. 2315	7	7
Or. 2420	7	7
Or. 2449	7	7
Oberschlesien	7	7
Phelps	7	7
Pocahontas	7	7
Premier	7	7
Red Crop	7	7
Red Rich Everbearing	7	7
Robinson	7	7
Sierra	7	7
Superfection	7	7
Surecrop	7	7
Talisman	7	7
Temple	7	7
Tennessee Beauty	7	7
Valentine	7	7
Vermillion	7	7
Wadenswil 3	7	7
Ydun	7	7
Agassiz	7	6
Ag. 88 ((Brit. Sov. x Or. 1639)S.)	7	6
Ag. 99 (Temple $S._2$)	6	7
Ag. 102 (Temple $S._2$)	7	6
Aroma Everbearing	6	7
Blakemore	6	7
Catskill	7	6
Cavalier	7	6
Dixieland	6	7
F. bracteata	7	6
F. virginiana	6	7
Guardsman	6	7
Macherauchs Frühernte	6	7
N.Y. 96	6	7
Pathfinder	6	7
Perle de Prague	6	7
Premier	7	6
S-4607	7	6
Ag. 72B (Or. 1639 S.)	5	7
Ag. 96 (Temple $S._2$)	6	6
Ag. 97 (Temple $S._2$)	6	6
Ag. 98 (Temple $S._2$)	6	6
Fairfax	6	6
Fairland	6	6
F. moschata	6	6
Madam Lefeber	6	6
Madame Moutot	6	6
Northwest	6	6
Stelemaster	6	6
Ag. 95 (Temple $S._2$)	5	6
Armore	5	6
Eden	5	6
Grenadier	5	6
Plentiful	5	6
Redglow	5	6
Deutsch Evern	5	5
Landia	5	5

CANADA DEPARTMENT OF AGRICULTURE, EXPERIMENTAL FARM, AGASSIZ, BRITISH COLUMBIA

Table 2. Average relative resistance to powdery mildew, with numbers and percentages of seedlings free from the disease, for populations of 27 strawberry crosses.

Cross	Number of seedlings in population	Average resistance rating	Number of seedlings free from the disease after fifth examination	Percentage rated 10
British Sovereign x F. chiloensis (Oregon)	446	8.16	0	
British Sovereign x F. chiloensis (Vancouver Island)	760	8.28	0	
British Sovereign x Or. 2467	358	8.37	2	.56
Or. 2467 x British Sovereign	10	8.40	0	
British Sovereign x F. ovalis	400	8.52	1	.25
British Sovereign x F. virginiana	677	8.09	5	.74
Agassiz x F. chiloensis (Oregon)	191	8.32	0	
Agassiz x Or. 2467	243	8.34	2	.82
Agassiz x Stelemaster	76	7.71	0	
Stelemaster x Agassiz	42	8.14	0	
Cavalier x Stelemaster	115	7.21	0	
Stelemaster x Cavalier	26	7.73	0	
Northwest x MD 683	72	7.99	0	
MD 683 x Northwest	21	7.05	0	
Northwest x Or. 2467	65	8.14	0	
Northwest x Pathfinder	395	7.81	0	
Pathfinder x Northwest	197	7.95	0	
Northwest x S-4607-9	93	7.05	0	
S-4607-9 x Northwest	413	8.18	0	
Northwest x Stelemaster	15	7.40	0	
Stelemaster x Northwest	30	7.87	0	
Puget Beauty x Siletz	121	8.97	28	23.14
Siletz x Puget Beauty	748	9.02	268	35.82
Redglow x Ag. 88	42	7.50	0	
Ag. 88 x Sparkle	135	7.90	0	
Sparkle x Ag. 88	66	8.32	0	

1959 WHEAT STREAK MOSAIC EPIPHYTOTIC IN KANSAS[1]

Claude L. King and Webster H. Sill, Jr.[2]

The severest outbreak of wheat streak mosaic virus ever recorded in Kansas occurred in winter wheat during the 1958-59 growing season. Several factors known to favor the development of the disease were present, both before and during the growing season, which may account for the disastrous epiphytotic.

The wheat-growing conditions for the 1957-58 crop were excellent with very little abandonment of wheat acreage, although there was considerable lodging owing to wet weather and high winds. The average yield was one of the highest on record, 27 bushels per acre. As expected under those conditions, there was an abundance of shattered seed on the ground during and after harvest. The seed germinated rapidly with adequate rain in June and July and produced much rank volunteer wheat in central and western Kansas, much of which grew throughout the summer and early fall.

The mite vector (Aceria tulipae Keifer (3)) populations increased rapidly throughout the summer on this volunteer wheat and by fall routine vector tests showed that many, if not most, of the mites tested were viruliferous. Since there was adequate moisture, most farmers in central and western Kansas planted wheat early, in late August or September, not only to take advantage of the moisture but also to obtain fall pasture. The fall proved to be long and unseasonably warm. This made it possible for mites to propagate and feed much later than usual, as warm temperatures favor reproduction and active feeding (2). By late fall the mite populations on much of the planted wheat had also become astronomical.

The combination of all these coincidental factors helped to make the disease outbreak practically inevitable and had it not been for the fact that approximately 40 percent of the Kansas wheat acreage is now grown to mosaic-tolerant varieties, Bison, Kiowa, Triumph and Concho (1), the losses would have been much greater. In severely diseased wheat fields Bison and Kiowa usually yielded about twice that of susceptible varieties such as Ponca and Wichita. Often the yield differences were even greater.

Loss estimates were made with the help of the county agricultural agents of Kansas and other agricultural specialists. Individual county loss estimates are given in Figure 1. Approximately 3,850,000 acres of wheat were infected in the State. Yield loss was estimated at 46,670,000 bushels, or approximately 20 percent of the total State wheat crop for the year.

Literature Cited

1. BELLINGHAM, R. C., HURLEY FELLOWS, and WEBSTER H. SILL, Jr. 1957. Resistance to wheat streak mosaic virus in foreign and domestic wheats and various wheat crosses. Phytopathology (Abst.) 47: 516.
2. DEL ROSARIO, MARIA SALOME E. 1959. Studies of Aceria tulipae Keifer (Eriophyidae) and other eriophyid mites in Kansas in relation to the transmission of wheat streak mosaic virus. Unpublished Ph.D. dissertation. Kansas State University of Agriculture and Applied Science, Manhattan.
3. SLYKHUIS, J. T. 1953. Wheat streak mosaic in Alberta and factors related to its spread. Canad. J. Agric. Sci. 33: 195-197.

[1] Contribution No. 665, Department of Botany and Plant Pathology, Kansas Agricultural Experiment Station, Manhattan. Department Serial No. 709.
[2] Extension Plant Pathologist and Plant Pathologist, respectively.

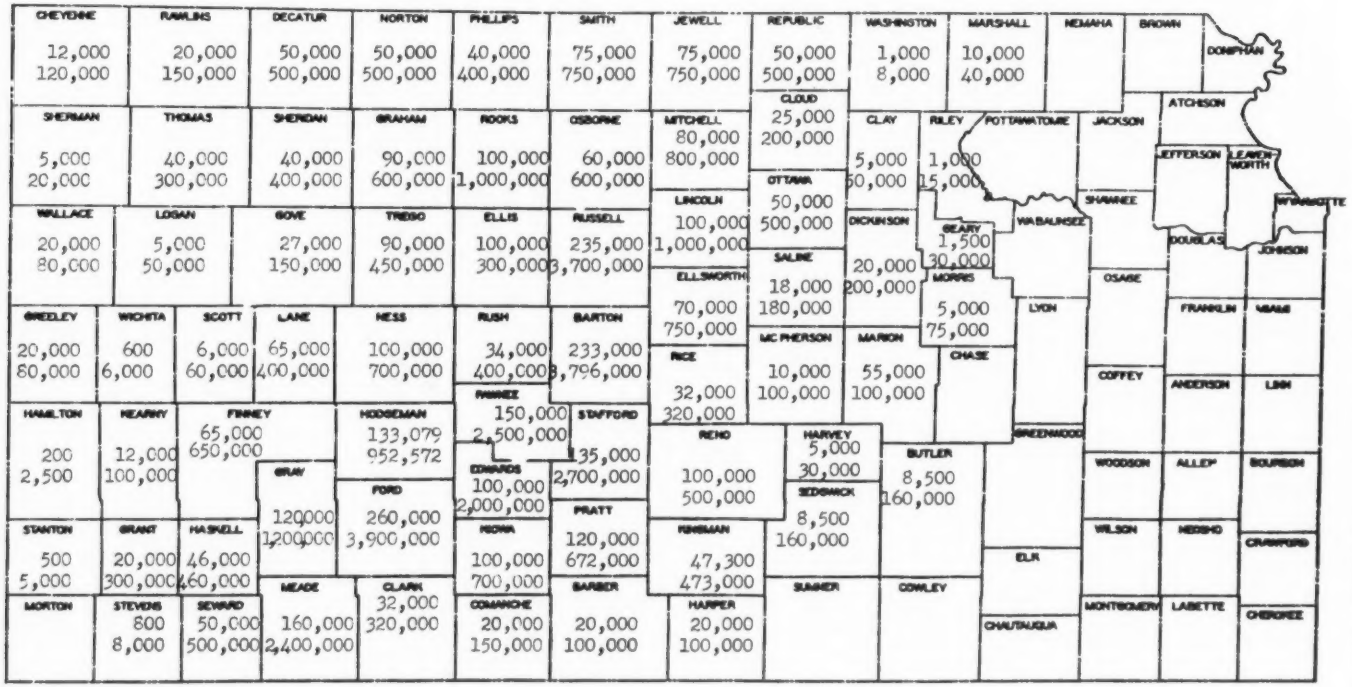


FIGURE 1. County losses due to wheat streak mosaic in Kansas: 1959 crop. Total Loss 46,673,000 bushels.

Top figure in each county is acres infected. Lower figure is loss in bushels.

HOST-PARASITE RELATIONSHIPS OF VARIOUS PLANTS AND THE SUGAR-BEET NEMATODE (HETERODERA SCHACHTII)[1]

A. Morgan Golden and Thelma Shafer[2]

Summary

In laboratory and greenhouse tests, 48 plant collections, representing 37 species of crop plants and weeds, were tested for susceptibility to the sugar-beet nematode in comparison with susceptibility of sugar beet. Eight of the species found to be susceptible apparently have not been previously reported as hosts for Heterodera schachtii.

Of 12 plant collections and species examined in addition to the sugar-beet control, root diffusates of nine significantly increased larval emergence from sugar-beet nematode cysts as compared with tap water. Although only one of these plants was approximately as effective as sugar beet, the results indicate that all nine plants produce a substance or substances which stimulate emergence of larvae from cysts of the sugar-beet nematode.

In another test, larvae of H. schachtii entered roots and developed at least to some degree in eight plant collections and species tested in comparison with sugar beet; mature males were found on two non-host plants in this test. Since bean (Phaseolus vulgaris) had a marked effect on emergence of larvae from cysts and was also invaded by large numbers of larvae, this plant may be of special value in nematode-reducing rotations.

INTRODUCTION

The present work was initiated in an effort to obtain a better understanding of the relationships existing between the sugar-beet nematode (Heterodera schachtii Schmidt, 1871), some of the crop plants which might be grown in rotation with sugar beets (Beta vulgaris), and some weeds which may be found in sugar beet fields. Among the weeds, several species of the genera Amaranthus and Chenopodium are given particular attention because of their widespread occurrence in beet-producing areas of the United States. Studies of this nature might clarify and extend the known host range of H. schachtii and also indicate which plants, if any, have possibilities as stimulatory-trap plants for use in reducing field populations of the sugar-beet nematode. The specific objectives were: 1) to determine the susceptibility to the sugar-beet nematode of the various plants specified in Table 1; 2) to measure the relative effectiveness of water and root diffusates of several plants (Table 2) on emergence of larvae from H. schachtii cysts; and 3) to ascertain how many larvae enter the roots of certain plants (Table 3) and to study their development after entering the roots.

MATERIALS AND METHODS

Susceptibility Tests

For each test plant species or selection in each experiment, 25 young plants from seeds germinated in sterilized sand were grown in the greenhouse for about 2 months in individual aluminum-foil cylinders containing soil heavily infested with H. schachtii cysts. This procedure, as well as the method of examination of the plants and of rating the infection, was essentially the same as described by Golden and Shafer (4). When white female nematodes or cysts were not seen on the roots of a particular test plant during the routine examination, the soil and plant roots from several cylinders were washed and sieved to recover any white females or new cysts which might have been overlooked.

---

[1] Cooperative research by the Crops Research Division, Agricultural Research Service, United States Department of Agriculture and the Beet Sugar Development Foundation.

[2] Respectively, Nematologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Beltsville, Maryland; and former Assistant Nematologist, Beet Sugar Development Foundation, Salinas, California.

Table 1. Susceptibility of various crop plants and weeds to the sugar-beet nematode.

Test plants	Common name	Source of seeds	Infection index rating[a] Experiment 1[b]	Experiment 2[b]
Allium cepa L.	onion	Salinas, Calif.	0	---
Amaranthus blitoides Wats.	--	Salinas, Calif.	0	0
A. carneus Greene	--	Salinas, Calif.	0.2	---
A. deflexus L.	--	Salinas, Calif.	0.1	---
A. graecizans L.	tumbleweed	Kearns, Utah	0	0
A. hybridus L.	green amaranth	Salt Lake City, Utah	0.2	---
A. palmeri S. Wats.	--	Calexico, Calif.	2.2	---
A. powelli S. Wats.	--	Salinas, Calif.	0	---
A. retroflexus L.	pigweed	Beltsville, Md.	0.3	0.5
A. retroflexus	pigweed	Lehi, Utah	0.4	0.7
A. retroflexus	pigweed	Payson, Utah	0.3	---
A. retroflexus	pigweed	Raleigh, N. C.	0.4	0.2
A. retroflexus	pigweed	Rocky Ford, Colo.	0.2	---
A. spinosus L.	spiny amaranth	Auburn, Ala.	0	0
A. tricolor L.	Joseph's-coat	Salinas, Calif.	0.3	0.6
Alyssum maritimum (L.) Lam.	sweet alyssum (carpet of snow)	Salinas, Calif.	0.2	0.6
A. maritimum	sweet alyssum (violet queen)	Salinas, Calif.	0.4	---
Apium graveolens L.	celery	Salinas, Calif.	0	---
Asparagus officinalis L.	asparagus	Salinas, Calif.	0	---
Beta vulgaris L.	sugar beet	Salinas, Calif.	4.0	4.0
B. vulgaris	swiss chard	Salinas, Calif.	4.0	---
Brassica campestris L.	common mustard	Salinas, Calif.	4.0	---
B. napus L.	turnip	Salinas, Calif.	4.0	---
B. oleracea L.	broccoli	Salinas, Calif.	4.0	---
B. oleracea	brussel sprouts	Salinas, Calif.	4.0	---
B. oleracea	cabbage	Salinas, Calif.	4.0	---
B. oleracea	cauliflower	Salinas, Calif.	4.0	---
B. oleracea	collards	Salinas, Calif.	4.0	---
B. pekinensis (Lour.) Rupr.	chinese cabbage	Salinas, Calif.	4.0	---
Chenopodium album L.	lamb's-quarters	Beltsville, Md.	0	0
C. album	lamb's-quarters	Raleigh, N. C.	0	0
C. ambrosioides var. chilensis (Schrad.) Spegaz.	--	Auburn, Ala.	2.2	2.0
C. capitatum (L.) Aschers.	strawberry-blite	Salinas, Calif.	2.9	--
C. schraderianum Roem. & Schult.	--	--	2.0	--
C. murale L. (collection A)	sowbane	Calexico, Calif.	3.2	2.8
C. murale (collection B)	sowbane	Calexico, Calif.	2.9	3.1
Daucus carota L.	carrot	Salinas, Calif.	0	--
Glycine max (L.) Merr.	soybean	--	0	--
Hesperis matronalis L.	dame's-violet	Chicago, Ill.	0	0
Lactuca sativa L.	lettuce	Salinas, Calif.	0	--
Linaria vulgaris Hill	butter-and-eggs	Salinas, Calif.	1.5	--
Malva parviflora L.	cheeseweed	Salinas, Calif.	0	--
Matthiola incana (L.) R. Br.	stock	Salinas, Calif.	0	--
Medicago sativa L.	alfalfa	Salinas, Calif.	0	--
Phaseolus vulgaris L.	navy beans	Salinas, Calif.	0	--
Raphanus sativus L.	radish	Salinas, Calif.	3.1	--
Rheum rhaponticum	rhubarb	Salinas, Calif.	4.0	--
Sonchus oleraceus L.	common sow-thistle	Salinas, Calif.	0	--
Spinacia oleracea L.	spinach	Salinas, Calif.	4.0	--

[a] Scale: 0 = no infection (no white females or new cysts found)
1 = trace (one or only very few white females or new cysts found)
2 = light (white females or new cysts in small numbers)
3 = moderate (white females or new cysts numerous)
4 = heavy (white females or new cysts very numerous)

[b] Infection index rating for each test plant represents an average rating of 25 plants (replications) in each experiment.

Table 2. Emergence of larvae from H. schachtii cysts in tap water or root diffusates of various crop plants and weeds in 3 separate experiments continued for 6 weeks.

Test plants[a]	Common name	Source of seeds	Emerged larvae total	Emerged larvae per cyst
Experiment 1				
Amaranthus retroflexus L.	pigweed	Rocky Ford, Colo.	21,820	136.4**
A. retroflexus	pigweed	Lehi, Utah	19,660	122.9*
A. retroflexus	pigweed	Beltsville, Md.	24,566	153.5**
Apium graveolens L.	celery	Salinas, Calif.	20,539	128.4*
Chenopodium album L.	lamb's quarters	Beltsville, Md.	22,700	141.9**
C. ambrosioides var. chilensis (Schrad.) Spegaz.	--	Auburn, Ala.	36,821	230.1**
Lactuca sativa L.	lettuce	Salinas, Calif.	13,001	81.2
Phaseolus vulgaris L.	navy bean	Salinas, Calif.	22,905	143.1**
Beta vulgaris L. (control)	sugar beet	Salinas, Calif.	41,109	256.9**
Tap Water (control)	--	--	11,494	71.8
Experiment 2				
Apium graveolens L.	celery	Salinas, Calif.	20,232	126.4*
Chenopodium album L.	lamb's quarters	Beltsville, Md.	19,226	120.2*
Phaseolus vulgaris L.	navy bean	Salinas, Calif.	23,655	147.8**
Beta vulgaris L. (control)	sugar beet	Salinas, Calif.	49,568	309.8**
Tap Water (control)	--	--	12,586	78.7
Experiment 3				
Malva parviflora L.	cheeseweed	Salinas, Calif.	21,808	136.4**
Linaria vulgaris Hill	butter-and-eggs	Salinas, Calif.	12,085	75.5
Lycopersicon esculentum Mill	tomato	Salinas, Calif.	13,257	82.9
Sonchus oleraceus L.	common sow-thistle	Salinas, Calif.	17,161	107.3*
Beta vulgaris (control)	sugar beet	Salinas, Calif.	48,220	301.4**
Tap Water (control)	--	--	11,275	70.5

[a] For each test plant, and each control, 160 selected cysts divided into 4 replications of 40 cysts each were used.

* Significantly greater than emergence in water at the 5 percent level.

** Significantly greater than emergence in water at the 1 percent level.

Table 3. Entrance and development of H. schachtii larvae in roots of various crop plants and weeds grown in cyst-infested soil for 15 and 30 days.

Exposure periods and test plants	Source of seeds	Nematodes[a] Per plant	Nematodes[a] Per gram of root	Avg. no. of males per plant	Stage of development[b]
15-day					
Amaranthus retroflexus	Rocky Ford, Colo.	57.8	75.4	0	2; 3
A. retroflexus	Lehi, Utah	118.6	77.3	0	2; 3
A. retroflexus	Beltsville, Md.	20.1	34.6	0	2; 3
Apium graveolens (celery)	Salinas, Calif.	0	0	0	--
Chenopodium album	Beltsville, Md.	17.1	15.1	0	2; 3
C. ambrosioides var. chilensis (Schrad.) Spegaz.	Auburn, Ala.	8.5	51.0	0	2; 3
Lactuca sativa (lettuce)	Salinas, Calif.	0	0	0	--
Phaseolus vulgaris (navy bean)	Salinas, Calif.	1.6	0.9	0	2
Beta vulgaris (sugar beet)	Salinas, Calif.	42.8	197.7	0	2; 3
30-day					
A. retroflexus	Rocky Ford, Colo.	642.0	126.3	11.0	2; 3; 4; 5M
A. retroflexus	Lehi, Utah	625.6	34.5	38.8	2; 3; 4; 5M;5F
A. retroflexus	Beltsville, Md.	772.5	50.1	2.5	2; 3; 4; 5M
A. graveolens	Salinas, Calif.	41.5	39.5	0	2; few 3's
C. album	Beltsville, Md.	865.0	135.1	10.8	2; 3; 5M
C. ambrosioides var.chilensis	Auburn, Ala.	1376.1	408.7	20.6	2; 3; 4; 5M;5F
L. sativa	Salinas, Calif.	13.3	1.4	0	2; few 3's
P. vulgaris	Salinas, Calif.	407.5	66.9	5.0	2; 3; 5M
B. vulgaris	Salinas, Calif.	2430.8	697.8	162.5	2; 3; 4; 5M; 5F

[a] Average of 6 plants (replications) for each species tested in each exposure period.

[b] 2 = second-stage (infective) larva; 3 = third-stage larva; 4 = fourth-stage larva; 5 = mature specimen; M = male; F = white gravid female.

Larval Emergence Tests

The methods and procedures were the same as used by Golden (3) in other larval emergence tests. Very briefly, for each test plant or control in each of the three experiments, 160 fresh, selected cysts divided into four replications of 40 cysts each were used. Each experiment was continued for 6 weeks. The tap water and root diffusates were changed weekly and the emerged larvae were removed and counted.

Larval Penetration and Development Tests

The procedures and techniques were similar to those used previously by Golden (3) except that the number of males per plant reported is the number found only in the roots of the test plants. The soil from the pots was not examined. Briefly, 12 young plants of each test plant were placed individually in 6-inch pots containing soil heavily infested with cysts of the sugar-beet nematode. After a 15-day growth period in the greenhouse, one-half of the plants were removed and the number and development of nematodes in the roots of each plant determined; after 30 days of growth the remaining half of the plants were examined in the same manner.

RESULTS AND DISCUSSION

Susceptibility to the sugar-beet nematode of the various crop plants (Table 1) is in agreement with results of Jones (7) and Winslow (12). In the present case, an attempt was made to show the relative degree of susceptibility of these plants as compared with susceptibility of sugar beet, which is desirable, but which was not reported by these authors. Several plants that apparently have not been previously reported as tested against the sugar-beet nematode were found susceptible, at least to some degree: Amaranthus carneus, A. deflexus, A. hybridus, A. palmeri, A. tricolor, Chenopodium ambrosioides var. chilensis, C. schraderianum, and Linaria vulgaris. Amaranthus graecizans was reported by Raski (8) as a host of H. schachtii but in both of the present tests the results with this plant were negative. The infection index rating of A. retroflexus, although low, appears to be reasonably consistent even though the seeds of the test plants came from different areas of the United States. These findings support the reports of Raski (8) and Corder, Buhrer and Thorne (1) that A. retroflexus is a host for the sugar-beet nematode, but conflict with the negative results obtained by Winslow (12). However, it may well be that either the plant, the nematode, or both are slightly different in England, where Winslow (12) did his work. Chenopodium album has been reported as a host for H. schachtii by several authors (1, 2, 9, 10, 12) and recently Golden and Shafer (4) showed that there are both resistant and susceptible races of this species. Evidently the two collections tested in the present study belong to the resistant race.

Results of the larval emergence tests (Table 2) indicate that all of the plants tested, with the exception of lettuce, tomato, and butter-and-eggs, produce a substance or substances which tend to stimulate emergence of larvae from cysts of the sugar-beet nematode. However, only Chenopodium ambrosioides var. chilensis approached sugar beet in its effect on emergence, while the other effective plants were intermediate between sugar beet and tap water. Comparison of the plant's susceptibility to H. schachtii and effectiveness upon larval emergence reveals a whole spectrum of relationships. On the one hand, there are host plants which, upon larval emergence, are: (a) very effective, as sugar beet; (b) intermediate, as pigweed; and (c) not effective, as butter-and-eggs and tomato (5). On the other hand, there are non-host plants which, upon larval emergence, also are: (a) very effective, as certain wild, resistant Beta species which were as effective as sugar beet (3); (b) intermediate, as navy bean and celery; and (c) not effective, as lettuce. The question arises of whether the stimulatory material apparently in diffusates of these and various other effective plants (11) is physically and chemically identical with the "hatching factor" in diffusate of sugar beet.

The larval penetration and development studies (Table 3) show that some larvae entered the roots of all test plants during the 30-day exposure period and that at least limited development occurred. However, the number of larvae which entered the roots of sugar beet and C. ambrosioides var. chilensis (both good hosts) was much greater at the 30-day exposure than the number found in the roots of any of the other plants. In bean and Chenopodium album (both non-hosts) males were found in small numbers. Only a very few larvae were found in lettuce, while fair numbers of larvae entered beans. In recent studies of six rotation systems involving the sugar-beet nematode, Johnson and Wheatley (6) found that the highest yields of sugar beets were obtained in the two rotations having beans as the immediate preceding crop.

One of the factors contributing to this beneficial effect might be the stimulatory-trap crop effect noted in these experiments, and it is suggested that beans might have some practical value in rotation systems for the control of the sugar-beet nematode.

Literature Cited

1. CORDER, M. N., E. M. BUHRER, and G. THORNE. 1936. A list of plants attacked by the sugar-beet nematode (Heterodera schachtii). Plant Disease Reptr. 20: 38-47.
2. FRANKLIN, M. T. 1951. The cyst-forming species of Heterodera. Commonwealth Agricultural Bureaux, Farnham Royal, Bucks., England. 147 pp.
3. GOLDEN, A. M. 1958. Interrelationships of certain Beta species and Heterodera schachtii, the sugar-beet nematode. Plant Disease Reptr. 42: 1157-1162.
4. GOLDEN, A. M., and THELMA SHAFER. 1958. Differential response of Heterodera schachtii, the sugar-beet nematode, to selections of Chenopodium album. Plant Disease Reptr. 42: 184-187.
5. GOLDEN, A. M., and THELMA SHAFER. 1959. Susceptibility of tomato (Lycopersicon esculentum) to the sugar-beet nematode (Heterodera schachtii). Plant Disease Reptr. 43: 1196-1197.
6. JOHNSON, R. T., and G. W. WHEATLEY. 1959. The effects of different rotations on sugar beet production in land infested with the sugar-beet nematode, Heterodera schachtii, in the Salinas Valley of California. J. Am. Soc. Sugar Beet Tech. 10: 286-289.
7. JONES, F. G. W. 1950. Observations on the beet eelworm and other cyst-forming species of Heterodera. Ann. Appl. Biol. 37: 407-440.
8. RASKI, D. J. 1952. On the host range of the sugar-beet nematode in California. Plant Disease Reptr. 36: 5-7.
9. THORNE, G. 1922. The sugar-beet nematode in the western states. U. S. Dept. of Agr. Farmer's Bull. 1248. 16 pp.
10. THORNE, G. 1952. Control of the sugar-beet nematode. U. S. Dept. of Agr. Farmer's Bull. 2054. 18 pp.
11. WINSLOW, R. D. 1954. The hatching responses of some root eelworms of the genus Heterodera. Ann. Appl. Biol. 43: 19-36.
12. WINSLOW, R. D. 1954. Provisional lists of host plants of some root eelworms (Heterodera spp.) Ann. Appl. Biol. 41: 591-605.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE AND BEET SUGAR DEVELOPMENT FOUNDATION, SALINAS, CALIFORNIA

COMPARATIVE TREATMENTS FOR POWDERY MILDEW OF APPLE[1]

L. E. Dickens[2] and W. J. Henderson[3]

Powdery mildew of apple (Podosphaera leucotricha (Ell. & Ev.) Salm.) was very destructive in apple-growing districts, especially in Delta County, Colorado, during the season of 1958. Certain orchardists obtained excellent control, while others reported unsatisfactory results with the standard recommended Karathane and micronized wettable sulfur. In order to demonstrate and to test further the efficacy of these control measures, spray plots were selected and established in a commercial orchard at Paonia, Colorado in the season of 1959. The orchard was selected on the basis of uniformity in size, age of trees, and prevalence of powdery mildew.

The experimental design consisted of five randomized complete blocks (replications) with three trees per treatment in each block. The chemicals used in the test were Karathane WD, micronized wettable sulfur, and Phytoactin L 307. Each chemical was suspended in 100 gallons of water. Triton B-1956 spreader was added, except for the Phytoactin spray. Karathane was applied at the rate of 3/4 pound per 100 gallons of water, sulfur at 6 pounds per 100 gallons of water, and Phytoactin at 200 ppm in 100 gallons of water. All trees received approximately the same volume of spray material. Three replications were of the Jonathan variety and two replications were of the Rome variety. Three applications of all materials were made: the first at the cluster-bud stage on May 6, the second at the calyx stage on May 20, and the first cover on June 2. Two additional cover sprays were applied on June 16 and July 4 to plots receiving Karathane. The sprays were carefully applied at the time of day when drift was at a minimum. The consistency of the data showed no detectable influence of drift.

Readings were taken on July 9. The number of visibly infected twigs having a length of 1 foot or more were counted on the west half of the center tree of each replication. There were no apparent phytotoxic effects of the chemicals. Difference in total number of mildew-infected shoots was quite obvious among the treatments. The data for the respective treatment replications were uniform. Control of powdery mildew by chemical treatment is summarized in Table 1.

Table 1. Control of powdery mildew of apple by chemical treatment.

Chemical	Infected shoots[a]	Percent control[b]
Sulfur	31.60[c]	87.6
Karathane WD	46.40	81.8
Phytoactin L 307	90.40	74.5
Control	255.00	0.0

[a]Average number of mildew-infected shoots per half tree for five replications.

[b]Percentage reduction in prevalence of mildew-infected shoots as compared with non-treated control.

[c]Significantly different treatment means at .05 level of probability are not scored by the same line when compared by least significant ranges, using Duncan's Multiple Range Test.

Statistical differences among treatments were highly significant, with no significant differences among replications. The two apple varieties appeared to be equally susceptible to powdery mildew.

Reduction in incidence of apple powdery mildew obtained with Karathane and micronized wettable sulfur sprays was outstanding. Phytoactin showed some promise, but it was less effective than either Karathane or the sulfur.

The cost per application is approximately the same for Karathane and sulfur. The difference in their effectiveness in reducing the incidence of apple powdery mildew is not appreciable. Although the sulfur was the most effective in controlling powdery mildew, it causes russeting of fruit during periods of high temperatures and for this reason it cannot be used as a cover spray.

It is the authors' opinion that excellent control of apple powdery mildew will be obtained with Karathane or sulfur when properly applied according to current recommendations of not more than 14-day intervals, beginning at the cluster-bud stage.

DEPARTMENT OF BOTANY AND PLANT PATHOLOGY,
COLORADO STATE UNIVERSITY, FORT COLLINS, COLORADO

[1] Published with the approval of the Director, Colorado Agricultural Experiment Station as Scientific Series Paper No. 631.

[2] Assistant Professor and Assistant Plant Pathologist, Department of Botany and Plant Pathology, Colorado State University.

[3] Extension Plant Pathologist, Colorado State University.

SUMMARY OF PLANT DISEASES IN VIRGINIA, 1959

S. B. Fenne[1]

FIELD CROPS

Corn: Corn diseases were severe during the seedling stage. Many farmers plant corn in direct contact with fertilizer, applied in the row. When normal rainfall occurs this practice suffices; however the past season was extremely dry at the time corn was planted and for about 3 weeks afterward. As a result seedlings were severely injured, primarily by FERTILIZER BURN, which permitted the entry of soil organisms causing SEEDLING BLIGHT. BACTERIAL WILT, SMUT, NUTRIENT DEFICIENCY and STALK ROTS were also reported.

Barley: SCAB was the most destructive disease of barley and in many cases caused considerable shriveling of grain and lower yields. SCALD was somewhat more common than usual. YELLOW DWARF was reported.

Oats: Unfavorable growing conditions during the first part of the year, coupled with nutritional deficiencies, caused very poor growth of oats. FOOT ROT and RED LEAF were reported.

Wheat: This season was a most favorable one for SCAB. Almost every wheat field showed some injury, and in a few cases yields were reduced by one-half. GLUME BLOTCH was quite prevalent; however it is doubtful whether glume blotch reduced yields greatly. LEAF RUST and STEM RUST were about average in occurrence. SOOTY MOLD continued to be reported by farmers who observed the blackened condition of their grain at about maturity and assumed that sooty mold was the cause of the shriveled grain. It is difficult to convince farmers that sooty mold is merely a saprophyte. In one county, two farmers (brothers) have been using the same seed for many years. As a result, about 50 percent of their plants were stunted and twisted because of NEMATODES. The TWIST DISEASE, caused by Dilophospora alopecuri, was found associated with the nematode infection.

Cotton: Considerable SEEDLING BLIGHT occurred early. Fertilizer injury and dry weather perhaps contributed largely to the amount of seedling blight. FUSARIUM WILT was observed and 2, 4-D INJURY was frequently reported.

Peanuts: STEM ROT was the disease most commonly found in peanuts. LEAFSPOT was not so severe as usual. Most farmers apply sulfur for leafspot control. NEMATODES are the cause of one of the most important diseases of peanuts.

Soybeans: There was some injury to soybeans from STEM ROT and NEMATODES. MOSAIC was reported in a number of cases.

Tobacco: BLUE MOLD appeared considerably later than usual on tobacco and caused very little injury. This is probably the result partly of (a) dry weather during the plant growing season and, (b) the use of control measures by most farmers. Tobacco was planted on time and there was an adequate supply of plants. Some ANTHRACNOSE was reported; however in such cases blue mold control sprays were not applied. PYTHIUM occurred early in the season. In some instances fields were replanted three and four times before suitable growing weather occurred and plants grew off. In several fields where Pythium appeared to be the cause of injury, a later check indicated that BLACKSHANK was present. Identification of blackshank has been considerably complicated by the development of resistant varieties. Frequently resistant varieties will show only limited symptoms of blackshank when compared with susceptible plants. With field tobacco the most spectacular problem was LIGHTNING INJURY. HOLLOW STALK continues to cause considerable consternation among farmers; however actual loss resulting is negligible. SUNSCALD frequently followed VIRUS INFECTIONS. The usual amounts of GRANVILLE WILT, NEMATODES, SORESHIN and NUTRIENT DEFICIENCY diseases were observed.

[1]Extension Plant Pathologist, Virginia Polytechnic Institute, Blacksburg, Virginia.

Alfalfa: STEM ROT was the most common disease of alfalfa early in the season. There were a number of cases of yellowing, which apparently were caused by a LACK OF NODULATION. In several areas alfalfa was planted during a dry season and apparently the inoculum died in the dry soil. There was sufficient moisture for seed germination and as long as nitrogen was available alfalfa grew quite well. When the nitrogen was used up the plants turned yellow and failed to grow. DOWNY MILDEW occurred frequently but serious injury did not result. ANTHRACNOSE and BLACKSTEM were more severe on alfalfa this year than for a number of years past. VARIOUS LEAFSPOTS were reported.

Clover: CROWN ROT and STEM ROT were the most common diseases on clover. Later, NORTHERN ANTHRACNOSE appeared; however SOUTHERN ANTHRACNOSE was much more severe later in the season. Several instances of VIRUS DISEASES and VARIOUS LEAFSPOTS were reported.

Grasses: Many complaints were received of injury to grasses in permanent pastures, but more often in lawns. VARIOUS FUNGI could be found on these grass and sod specimens, especially Helminthosporium, Rhizoctonia and Fusarium. However, it is believed that most of the injury was due to UNFAVORABLE GROWING CONDITIONS and POOR MANAGEMENT. In many cases the soil was dry and low in nutrients. Lawns were frequently clipped too short, and in some cases were injured by too much fertilizer.

TRUCK CROPS

Beans: ROOT ROT was the most common disease of garden beans. Several different organisms were associated with this root rot, which is always more severe when beans are planted in the same area several years in succession. During the summer when it was exceedingly hot and dry red spiders caused severe injury, which complicated the diagnosis of various diseases. MOSAIC and ANTHRACNOSE continue to be found commonly on garden beans.

Cantaloupes and Cucumbers: BACTERIAL WILT was the most common disease found on cantaloupes and cucumbers. ANTHRACNOSE was also frequently reported. NEMATODES are an ever-present problem, especially in gardens that have not been rotated.

Irish Potatoes: Only a trace of LATE BLIGHT was reported. There were several instances of BLACK-LEG and MOSAIC was occasionally reported.

Sweetpotatoes: BLUESTEM was the disease most often reported on sweetpotatoes. The usual amounts of SCURF and BLACK ROT were also observed.

Tomatoes: LATE BLIGHT was only present in a few areas, especially in far eastern Virginia and in the mountain areas. Losses were negligible. The diseases observed most were FUSARIUM WILT, MOSAIC, EARLY BLIGHT, GRAY MOLD, BACTERIAL SPOT, and one case of GHOST SPOT. NEMATODES were commonly present. Injury due to WEED KILLERS, such as 2,4-D, frequently complicated disease identification.

EXTENSION SERVICE, VIRGINIA POLYTECHNIC INSTITUTE, BLACKSBURG, VIRGINIA

THE CONTROL OF ERGOT IN CEREAL CROPS[1]

W. P. Campbell[2] and H. A. Freisen[3]

Summary

Through experiments carried out at Edmonton and Lacombe, Alberta it has been found that it may be possible to control ergot in headland grasses by spraying with maleic hydrazide at the rate of 8 pounds active ingredient per acre. The suppression of heading prevents the development of ergot sclerotia in the grasses of the headlands and eliminates the chief source of inoculum for the adjacent cereal crops.

INTRODUCTION

Grain fields are usually most severely infested with Claviceps purpurea (Fr.) Tul. near the edges, which suggests that most infection must originate from the borders of the fields. It has been established (1) that the wild grasses do, in fact, constitute a reservoir of ergot inoculum for our crop plants, and that the fungus passes readily from one host species to another. Thus, controlling infection of the wild hosts is of prime importance in the control of ergot in the field crops. As long as a century ago Kühn (2) recommended cutting the wild grass beside the fields and roads before it had reached a stage where ergot sclerotia could develop. However, in many cases the headlands are so obstructed with rocks, trees, ditches and fences that machine mowing is impractical and other methods of preventing the production of heads are needed. Herbicides are available that will kill the grass but their use is undesirable because of the weed and erosion problems that would arise if the grasses were removed. Thus, the answer seems to lie in the application of a chemical spray at a rate which will prevent heading of the grass but will not kill it, and of a type that can be applied with the spray equipment currently in use on the farms.

EXPERIMENTS AND RESULTS

Preliminary tests of the following four chemicals were conducted in the greenhouse and in field plots, using Bromus inermis as a test species: Dalapon (sodium $\alpha\alpha$ - dichloropropionate), Aminotriazole (3-amino- 1, 2, 4- triazole), Cyanamid (calcium cyanamide), and Maleic hydrazide (1, 2- dihydropuridazine -3, 6-dione). These herbicides were applied at rates varying from 0.08 pounds to 20 pounds per acre. It was found that Dalapon and Aminotriazole either killed the grass completely or did not control heading and the effects of Cyanamid were inconsistent. Maleic hydrazide, however, at 5, 10, 15 and 20 pounds per acre gave excellent control of heading without attendant killing of the plants. Enough water was used to cover the area of the test plot.

Maleic hydrazide was tested further in the greenhouse on Agropyron repens, A. cristatum, Festuca rubra, Poa pratensis, and Phleum pratense. Heading was suppressed by the 10-pound rate in all species tested.

Maleic hydrazide was applied to B. inermis in plots to determine the required rate of application. Plots were sprayed at the rates of 2, 4, 6, 8 and 10 pounds per acre. The two lowest rates delayed heading for a time but the other treatments prevented heading during the entire growing season.

A test using power spray equipment was conducted at the Experimental Farm, Lacombe, Alberta to determine the feasibility of chemical suppression of heading on a field scale. Maleic hydrazide (MH 30) was applied to roadside grasses (predominantly B. inermis) on June 5 at the rate of 8 pounds active ingredient per acre. When examined on August 12 the plants in the treated areas were still not headed while the unsprayed plants had matured seed.

---

[1] Joint contribution: Plant Pathology Laboratory and Experimental Farm, Research Branch, Canada Department of Agriculture, Edmonton and Lacombe, Alberta. Plant Pathology Laboratory Contribution No. 101.

[2] Plant Pathology Laboratory.

[3] Experimental Farm.

DISCUSSION

Ergot infection in cereals originates mainly from ascospores of C. purpurea released by sclerotia of the fungus in the grass headlands and ditches adjacent to the fields. Sclerotia are formed only in the floral parts of the host. Results of the present experiments show that grasses can be prevented from heading by a single application of maleic hydrazide. Furthermore, since this chemical is effective without killing the grass there is no tendency to promote soil erosion or the growth of weeds.

Literature Cited

1. CAMPBELL, W. P. 1957. Studies on ergot infection in gramineous hosts. Can. J. Botany 35: 315-320.
2. KÜHN, J. 1863. Untersuchungen über die Entstehung, das kühstliche Hervorrufen und die Verhütung des Mutterkorns. Landwirtsch. Instituts der Universitat Halle, 1-36.

CANADA DEPARTMENT OF AGRICULTURE, EDMONTON AND LACOMBE, ALBERTA

REACTION OF RESCUEGRASS VARIETIES AND STRAINS TO THE HEAD SMUT FUNGUS[1]

Howard W. Johnson[2]

Seed of seven varieties and strains of rescuegrass (Bromus catharticus) were inoculated with chlamydospores from five collections of the head smut fungus (Ustilago bullata Berk.) in November 1958. The same smut collections had been used in earlier inoculation tests[3]. Chlamydospores of each collection were obtained from smutted panicles of Chapel Hill rescuegrass grown at Stoneville, Mississippi in 1955-56. The smutted panicles had been stored in paper bags at 60° F until used in the 1958-59 test. Since no smut had developed in noninoculated rows of Chapel Hill rescuegrass in the 1955-56 test, the possibility of mixture through seed-borne inoculum did not exist, and it was assumed that the different collections of head smut were uncontaminated.

Smutted spikelets from each of the five collections were ground with a mortar and pestle to a fine black powder. These inocula were shaken separately with seed of the seven varieties and strains of rescuegrass until the seed were heavily coated with spores. The seed were planted at Stoneville on November 7, 1958 in four replications of a split-plot design with rescuegrass varieties as whole plots and fungus collections as split-plots. Each split-plot was a 10-foot row.

In May 1959 the panicles harvested from each row in the test were placed separately in large paper bags. The number of panicles harvested varied from 33 to 360 per row, but was usually between 100 and 200. The numbers of smutted and smut-free panicles were determined soon after harvest and the results were calculated as percentages of smutted panicles. The percentages were transformed to degrees and the transformed data were subjected to the analysis of variance[4].

Table 1. Percentage data from a rescuegrass head smut inoculation test made at Stoneville, Mississippi in 1958-59 transformed to degrees.

Variety or strain of rescuegrass	Degrees[a] of smutted panicles where seed were inoculated with spores from indicated head smut collection					
	Auburn, Ala.	Watkinsville, Ga.	Baton Rouge, La.	Raymond, Miss.	College Station, Texas	Variety or strain mean[b]
Commercial	64.1	66.0	65.0	49.1	60.6	60.6**
Chapel Hill	33.4	39.3	31.5	25.3	30.3	32.1**
Georgia Selection	0.0	3.1	4.8	0.0	2.6	2.6
Lamont	0.0	0.0	3.6	0.0	2.6	1.8
Lilimac	58.1	62.2	64.7	60.8	61.8	61.5**
Nakuru	6.0	4.4	0.0	9.3	10.3	7.0
Texas 46	70.9	73.4	74.0	76.7	76.7	74.2**
Collection mean[c]	38.7	40.5	39.6	36.6	39.0	--

[a] Each figure is the mean of four replicates, each consisting of a 10-foot row.
[b] Double asterisks mark the means that are significantly higher than Lamont at the 1-percent level. L.S.D. = 6.2 degrees.
[c] Differences between these means are not statistically significant. L.S.D. (5%) = 4.1 degrees, when based on the variety x collection mean square.
Coefficient of variation = 11.8 percent.

[1] A report of cooperative work conducted by the Crops Research Division, Agricultural Research Service, United States Department of Agriculture and the Delta Branch of the Mississippi Agricultural Experiment Station at Stoneville, Mississippi.
[2] Research Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and Mississippi Agricultural Experiment Station.
[3] Johnson, Howard W. 1957. Lamont, a new variety of rescuegrass resistant to head smut. Phytopathology 47: 409-412.
[4] Bliss, C. I. 1938. The transformation of percentages for use in the analysis of variance. Ohio J. Sci. 38: 9-12.

The seed used in this test were not treated with a fungicide before inoculation. Plants of the Chapel Hill, Georgia Selection, Lamont, and Nakuru varieties and strains from noninoculated seed remained smut-free. Plants of Commercial, Lilimac, and Texas 46 from noninoculated seed averaged 1.4, 1.6, and 24.3 percent smutted panicles, respectively. Therefore, some of the smut in inoculated rows of these strains, especially Texas, 46, doubtless came from natural seed-borne inoculum.

Commercial, Chapel Hill, Lilimac, and Texas 46 were susceptible to each of the five collections of U. bullata (Table 1). In contrast, Georgia Selection[5], Lamont, and Nakuru were resistant or only slightly susceptible. The means for the four susceptible varieties and strains were all significantly higher at the 1-percent level than was the mean for the Lamont variety. The means for Georgia Selection and Nakuru were not significantly higher than the mean for Lamont. However, only two smut collections infected the Lamont variety, while three caused slight smutting of Georgia Selection and four were able to cause smut on Nakuru. The differences between means for smut collections were not statistically significant when all varieties were combined.

Lamont rescuegrass was released as a head smut-resistant variety in 1957[3]. The results of this inoculation test show that it has retained its high resistance to several head smut collections from the southeastern United States. Georgia Selection and Nakuru also possess resistance to the head smut fungus, when tested against these collections.

DELTA BRANCH, MISSISSIPPI AGRICULTURAL EXPERIMENT STATION, STONEVILLE, MISSISSIPPI

[5] A selection made by J. M. Elrod, Georgia Experiment Station, Experiment, Georgia.

ADDITIONAL UNRECORDED HOSTS OF ARMILLARIA MELLEA

Robert D. Raabe[1]

The oak root fungus, Armillaria mellea (Fr.) Quél., causes considerable damage in home gardens in California. For this reason, in 1938 the University of California began searching for resistant plants by growing many species in a plot known to be infested with the fungus at the Deciduous Fruit Field Station at San Jose, and to which additional inoculum is added occasionally. To the revised list of hosts based on the first plantings (3) should be added the following hosts: Eucalyptus rudis Endl. (Desert Gum or Moitch), and Erica carnea L. (Spring Heath).

In the new plantings, started within the last 4 years, are additional species which are showing susceptibility to the fungus. These include Callistemon viminalis Cheel., Caryopteris clandonensis Simmonds, Cassia tomentosa L. f., Ceanothus thyrsiflorus Esch. (Blue-Blossom), Cercidiphyllum japonicum Sieb. & Zucc. (Katsura-Tree), Cercis canadensis L., Euphorbia pulcherrima Willd. (Poinsettia), Fremontia mexicana Macb., Hakea laurina R. Br. (Sea Urchin), Hibiscus syriacus L. (Rose-of-Sharon), Hypericum prolificum L., Kohlreuteria paniculata Laxm. (Goldenrain-Tree), Laburnum anagyroides Medic. (Golden-Chain, Bean Tree), Lithocarpus densiflorus Rehd. (Tanbark Oak), Malus hupehensis Rehd., M. sargentii Rehd., M. sylvestris Mill. var. Eleyi Bailey, M. toringoides Hughes, Melaleuca genistifolia Sm, Melaleuca leucadendra L. (Cajeput Tree, Punk-Tree), Paulownia tomentosa Steud., Poinciana pulcherrima L. (Barbados Pride, Barbados Flower-Fence, Dwarf Poinciana), Rhus trilobata Nutt. (Ill-scented Sumac), and Ulex europaeus, L.

In addition, a plant of Rhaphiolepis indica Lindl. (India-Hawthorn) from Orinda, California, a plant of Maytenus boaria Molina (Mayten) from Piedmont, California, and one plant each of Agave attenuata Salm-Dyck. and Eucalyptus maculata Hook. var. citriodora Bailey (E. citriodora Hook.) (Lemon-scented Spotted Gum) from Santa Barbara, California have been found infected.

It is believed that this is the first record of Armillaria mellea on these plants except for Poinciana pulcherrima, which was recorded as a host in Uganda in 1923 (6), Euphorbia pulcherrima, listed as a host in Australia (5), and Eucalyptus maculata var. citriodora which was recorded as a host in Tanganyika (7). Other possible duplications include Ceanothus thyrsiflorus (a Ceanothus sp. was listed as being infected in England (1)), Laburnum anagyroides (Beaumont (2) listed a Laburnum sp. as being infected), and Paulownia tomentosa (Moore (4) listed a Paulownia sp. as being a host).

Literature Cited

1. ANON. 1936. England & Wales: new and interesting phytopathological records for the year 1935. Int. Bull. Plant Prot. 10, 3: 49-50. (Rev. Appl. Mycol. 15: 478).
2. BEAUMONT, A. 1937. Fourteenth annual report of the Department of Plant Pathology. Seale-Hayne Agriculture College Pamphlet No. 48, 48 pp.
3. DEPARTMENT OF PLANT PATHOLOGY. 1956 (Revised). List of ornamentals tested for resistance to the oak root fungus (Armillaria mellea) and tentative rating of each based on tests at the University of California Deciduous Fruit Field Station at San Jose. University of California Agr. Ext. Mimeograph, 3 pp.
4. MOORE, W. C. 1943. Diseases of crop plants. Bull. No. 126 of the Ministry of Agriculture and Fisheries. 100 pp.
5. NOBLE, R. J., H. J. HYNES, F. C. McCLEERY, and W. A. BIRMINGHAM. 1934. Plant diseases recorded in New South Wales. Agr. Dept., New South Wales Science Bull. 46, 47 pp.
6. SMALL, W. 1923. Annual report of the government mycologist. Uganda Dept. Agr. Ann. Rept., pp. 14, 15. (Abst. in Experiment Station Record 53: 147-148).
7. WALLACE, G. B. 1935. Armillaria root rot in East Africa. The East African Agricultural J. 1: 182-192.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY, CALIFORNIA

[1]Assistant Professor of Plant Pathology, Department of Plant Pathology, University of California, Berkeley, California.

RELATION OF CROP SEQUENCE TO TASSEL SMUT OF CORN[1]

Roy D. Wilcoxson and R. P. Covey[2]

Corn smut caused by Ustilago maydis (DC.) Cda. is one of the prevalent diseases of dent corn in the United States. Because the disease can be controlled with resistant hybrids, little attention has been given to control by other means. The possible value of crop rotation for control of corn smut has been indicated by many people, but no one has suggested that crops other than corn might be responsible for increased incidence of smut in corn.

Such a relationship was observed in crop sequence plots established at the Agricultural Experiment Station, Rosemount, Minnesota. In this experiment two randomized strips each of corn, soybeans, oats, wheat, and flax are planted each year at right angles to similar strips from the preceding year, so that each crop succeeds each of the five crops (including itself) in four plots every year. The plots are approximately 40 feet square. Aside from the crops, the plots were managed as uniformly as possible and in a manner comparable to commercial practice. At different times during 1956-58, inclusive, the following corn hybrids and single crosses were planted in these plots: Minhybrid 507, Pioneer 349, W22xA73, Illinois 4226 x A334, and A322 x A334.

The percentages of plants with tassel smut just before harvest are shown in Table 1.

Table 1. Percentage of corn plants with tassel smut in plots previously planted with corn, wheat, soybeans, oats or flax.

Year	Previous crop and percentage of smut[a]				
	Corn	Wheat	Soybeans	Oats	Flax
1956	7	9	11	12	18
1957	12	12	21	20	19
1958	5	3	7	7	5
3 year Mean	8	8	13	13	14

[a] In 1956, 1957, and 1958 the percentages were based on a total of 600, 160, and 80 plants per plot, respectively.

All infection occurred naturally and nearly all of the smut occurred on the tassels. Infection of other plant parts was not related to previous crops or to other environmental factors.

The differences in percentages of plants with smut due to the influence of previous crops were not so apparent in 1958 as in 1957 and 1956, although the trend can be seen. In 1957 and 1956 more smut was found in plots previously planted to oats, soybeans, and flax than in those planted to corn or wheat. The 3-year averages of the data show that 8 percent of plants in plots previously planted with corn and wheat had smut while 13 or 14 percent of plants in plots previously planted with the other three crops had smut, a statistically significant difference ($P=.99$).

DEPARTMENT OF PLANT PATHOLOGY AND BOTANY, INSTITUTE OF AGRICULTURE, UNIVERSITY OF MINNESOTA, ST. PAUL, MINNESOTA

[1] Paper No. 4261, Scientific Journal Series, Minnesota Agricultural Experiment Station.

[2] Assistant Professor and Research Assistant, respectively, Department of Plant Pathology and Botany, University of Minnesota.

REDUCING AMARYLLIS LEAF SPOT BY SPRAYING[1]

D. L. Gill[2]

Summary

Amaryllis plants (Amaryllis sp.) were sprayed with fungicides from the small leaf stage until blooming for control of leaf spot (Stagonospora curtisii). Zineb, ferbam, captan, and Copper A all reduced infection. The first two gave greater reduction than the last two.

Amaryllis spp. are widely grown in the South. Leaf spot, or red-blotch, caused by Stagonospora curtisii (Berk.) Sacc. is widespread in the United States and is frequently considered of little importance (1, 4, 5, 6). The disease is of concern to southern growers, however, because of the characteristic spots on the leaves and also because the fungus attacks flower stalks and may cause them to topple before or during flowering or before the seed is mature. Many growers produce plants from seed, and these seedlings may remain in one bed for several years. Seedlings and other plants are commonly transplanted and sold in the South while blooming rather than when dormant. Because of this method of culture, bulb treatments are not feasible for control of this disease. Weiss (6) found a 1-200 formaldehyde soak for 2 hours beneficial. He suggested a mercuric chloride 1-1000 soak for 2 hours or soaking in water for 2 hours and then in either material for 30 minutes. Creager (2) and Haasis (3), working with narcissus infected by S. curtisii, found mercuric chloride 1-750 or formalin 1-120 for 2 hours or for 30 minutes after presoaking 1 hour in water beneficial. Some southern growers wish recommendations for control by means other than bulb treatment. No results of spraying amaryllis for control of Stagonospora curtisii are known, but Haasis (3) reported its control on narcissus by spraying with 4-4-50 Bordeaux mixture or dusting with 20-80 copper lime dust. Weiss (6) and Brierley (1) suggested the use of Bordeaux mixture with a sticker. Some results of spraying amaryllis for leaf spot control are reported here.

MATERIAL AND METHODS

A planting of seedling amaryllis which had been in place several years was divided into 35 plots in February 1956. Each plot consisted of three rows 2 1/2 feet apart and 25 feet long. Four spray treatments and an unsprayed control were used in each replication, and seven replications were used in the test. Materials used as sprays were captan 50 percent (2 pounds per 100 gallons), ferbam (2 pounds per 100 gallons), zineb (2 pounds per 100 gallons), and Copper A (4 pounds per 100 gallons). A sufficient amount of the detergent "Dreft" was added to each spray to allow thorough wetting of the foliage. The first application was made March 5, 1956 when the foliage was small. Further sprays were applied March 14 and 29 and April 10, 20, and 30. The test was repeated in 1957 when applications were made February 28, March 14 and 28, April 10 and 19, and May 2. Ratings of 75 leaves in the center row of each replication were made May 24, 1956 and May 19, 1957. Ratings were from 0 for no infection to 5 for three-quarters or more of a leaf infected. The sum of the 75 ratings was divided by 75 and multiplied by 20 to obtain the disease index.

RESULTS AND DISCUSSION

The means of the disease indices for the 2 years are shown in Table 1. All treatments significantly reduced infection, but zineb and ferbam gave better control than captan or Copper A. Results in the 2 years are similar. These data indicate that in the South amaryllis can be sprayed with zineb or ferbam at 10-day to 2-week intervals from the time foliage is small in

---

[1]Cooperative investigations at Tifton, Georgia of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and the University of Georgia, College of Agriculture, Agricultural Experiment Station. Published with the approval of the Director as Journal Series No. 71.

[2]Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Tifton, Georgia.

Table 1. Value of fungicides for control of amaryllis leaf spot (Stagonospora curtisii) at Tifton, Georgia, 1956 and 1957.

Treatment	Mean disease index	
	1956	1957
Zineb (2 pounds/100 gallons)	36.3	35.7
Ferbam (2 pounds/100 gallons)	39.9	39.3
Captan 50% (2 pounds/100 gallons)	44.6	45.4
Copper A (4 pounds/100 gallons)	49.0	52.3
None (check)	59.1	60.1
L.S.D. 5 percent	6.2	5.2
L.S.D. 1 percent	8.3	7.0

the spring until blooming to reduce the amount of leaf spot. Sufficient sticker-spreader must be added to the spray to obtain coverage. The spray should be allowed to run down into the base of the bulbs since infection is frequent on the bulb scales. As an aid in control, old plant debris should be removed before growth starts in the spring. Rotation should be followed when practical. In areas which allow air circulation less disease should develop.

Literature Cited

1. BRIERLEY, PHILIP. 1948. Diseases of Amaryllidaceae excluding those of Allium and Narcissus. Herbertia 15: 113-119.
2. CREAGER, D. B. 1933. Leaf scorch of narcissus. Phytopathology 23: 770-786.
3. HAASIS, F. A. 1934. Control of the narcissus leaf-scorch under Long Island conditions. (Abst.) Phytopathology 24: 9-10.
4. LASKARIS, THOMAS, and B. O. DODGE. 1941. Red-blotch of Hippeastrum. Bull. Torrey Botan. Club 68: 463-466.
5. SMITH, C. O. 1935. Inoculations of Stagonospora curtisii on the Amaryllidaceae in California. Phytopathology 25: 262-268.
6. WEISS, FREEMAN A. 1934. The red leaf spot or "Rust" of amaryllis. Yearbook, Amaryllis Society (Herbertia) 1: 92-94.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE AND UNIVERSITY OF GEORGIA AGRICULTURAL EXPERIMENT STATION, TIFTON, GEORGIA

EFFECTS OF SOAKING EASTER LILY BULBS IN PURATIZED AGRICULTURAL SPRAY OR DEMETON, OR IN COMBINED SOAKS, ON APHIDS, BLACK SCALE AND YIELD[1]

D. L. Gill[2]

Summary

Easter lily bulbs were soaked in water 1 hour, in demeton 1-500 1 hour, in Puratized Agricultural Spray 1-500 24 hours, and in three combinations of the Puratized Agricultural Spray-demeton treatments. Bulb yields were reduced by Puratized Agricultural Spray soak alone or in combinations with demeton. Demeton did not reduce the effect of Puratized Agricultural Spray upon black scale (Colletotrichum lilii). Aphids were controlled by the combinations.

Black scale of Easter lily bulbs caused by Colletotrichum lilii Plak. is controlled as shown by Lebeau (2, 3, 4) by soaking diseased bulbs for 24 hours in Puratized Agricultural Spray 1-500. Lebeau also found that bulbs to be planted in infested soil should be dusted or dipped in thiram prior to planting to prevent reinfection. Some lily growers use the black scale soak treatment regardless of whether or not black scale is present on the bulbs. Questions have been received concerning the safety of this practice. When a soak of demeton 1-500 for 1 hour prior to planting bulbs was found to control aphids carrying viruses producing necrotic fleck (1), information was desired also as to the effectiveness of combining these two treatments. The tests reported here supply data on the effects of these treatments, singly or combined, on yield, on black scale and on aphids.

METHODS AND RESULTS

The six bulb treatments used in these tests from the 1955-1956 season through the 1957-1958 season are listed in Table 1. When two consecutive treatments were used, bulbs were placed on a screen between treatments and the excess material was drained off before the bulbs were placed in the second soak.

To determine the effects of the treatments on the disease, lots of 17 Easter lily scales with black scale lesions were treated with each of the six treatments, washed, and plated on potato-dextrose agar according to the procedure of Lebeau (3). The numbers of scales from which the causal fungus grew were Puratized Agricultural Spray 1, demeton 8, Puratized Agricultural Spray-demeton mixture 1, demeton followed by Puratized Agricultural Spray 0, Puratized Agricultural Spray followed by demeton 0, and water 7. These findings indicate that demeton did not affect the eradicant action of Puratized Agricultural Spray on C. lilii.

Bulbs treated with the six soaks were grown in the field during the 1955-1956 season through the 1958-1959 season to determine the subsequent growth of the bulbs. Nine replications were used each year and the six treatments were randomized in each. The plantings were made in October and the bulbs were dug in August. Bulbs of the Georgia variety were used except in the first year, when mixed Creole and Georgia lilies were grown. Soil was Tifton sandy loam. Black scale is favored by a heavier soil with a higher water-holding capacity. Diseased bulbs were planted in 1955 and 1956, but little of the disease appeared on the bulbs when dug. Healthy bulbs were used in later plantings.

The bulbs planted in 1955 varied in size because of selection for disease and the results are not reported here. In 1956, 15 bulbs approximately 3 inches in circumference were planted in each plot. The following year 18 bulbs (three 6-7 inch, three 5-6 inch, and twelve 2 1/2-3 inch) were used. In 1958, 15 bulbs (three 6-7 inch, three 5-6 inch, and nine 2 1/2-3 inch) were planted per plot.

The means of the total yields and stands for each treatment for 1956-1957 through 1958-

[1]Cooperative investigations at Tifton, Georgia of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture and the University of Georgia, College of Agriculture, Agricultural Experiment Station. Published with the approval of the Director as Journal Series No. 72.

[2]Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Tifton, Georgia.

Table 1. Yield and stand of Easter lilies from bulbs soaked with Puratized Agricultural Spray or demeton, singly or combined.

Treatment	Mean yield (in grams)			Mean stand (number)		
	1956-57	1957-58	1958-59	1956-57	1957-58	1958-59
Puratized Agricultural Spray 1-500 (24 hours)	493	761	561	13.3	17.8	12.2
Demeton 1-500 (1 hour)	617	1122	1002	13.8	16.6	15.0
Puratized Agricultural Spray 1-500 plus demeton 1-500 (24 hours)	493	267	182	13.3	9.7	6.1
Demeton 1-500 (1 hour) followed by Puratized Agricultural Spray 1-500 (24 hours)	213	268	520	8.9	12.2	10.1
Puratized Agricultural Spray 1-500 (24 hours) followed by demeton 1-500 (1 hour)	367	965	752	12.3	15.2	13.0
Water (1 hour)	695	1137	1026	14.3	17.4	14.9
L.S.D.						
0.05	221	240	164	1.9	2.0	2.3
0.01	294	321	219	2.5	2.7	3.1

1959 are shown in Table 1. Water soaks of the bulbs gave highest yields in these tests followed by demeton. Puratized Agricultural Spray reduced yields. The combination Puratized Agricultural Spray plus demeton and the demeton soak followed by Puratized Agricultural Spray gave lowest yields. The other two treatments, Puratized Agricultural Spray alone and Puratized Agricultural Spray followed by demeton, produced intermediate yields. Stand of plants (Table 1) followed a similar trend. Shoots from bulbs treated with demeton or with water emerged sooner and the plants consistently appeared better than those given other treatments.

Counts of aphids on plants in the field gave inconclusive results as to the effects of Puratized Agricultural Spray on the control of aphids by demeton. To obtain further information, lots of five 5-6 inch bulbs previously given each of the six treatments were planted one to a No. 10 can and placed in the greenhouse on November 18, 1958. Three bulbs approximately 2 1/2 inches in circumference were also placed in five additional cans per treatment. Soil was 1/3 peat moss and 2/3 sifted Tifton sandy loam. Cans were randomized on the bench.

The bulbs treated with Puratized Agricultural Spray-demeton mixture produced no plants. Fewer plants emerged from the bulbs treated with demeton followed by Puratized Agricultural Spray than from those given the other treatments. This treatment also resulted in poorer plants. Aphids were observed in late January on plants from bulbs treated with water or with Puratized Agricultural Spray, but not on any treated with demeton. On February 2, 2 to 5 Aphis gossypii were placed on each uninfested plant. Further aphid transfers to uninfested plants were made February 16 and March 3. Aphids became very abundant on plants from bulbs not treated with demeton. Several examinations of the plants from bulbs soaked in any of the demeton treatments showed only a limited number of aphids, which did not persist. However, they did feed on the plants long enough to transmit viruses producing fleck. In this test it appears that Puratized Agricultural Spray had little or no effect on aphid control by demeton.

CONCLUSION

Since Puratized Agricultural Spray soak 1-500 for 24 hours reduced yields in these tests, it should not be used unless black scale is present on the bulbs. The demeton soak, used for control of aphids, combined with Puratized Agricultural Spray had no effect on black scale control. Yields from bulbs given combination treatments were as poor as those from bulbs given the Puratized Agricultural Spray alone. If Puratized Agricultural Spray is to be used as a bulb soak, it would appear safer to apply demeton to the plants as a spray. If a combination bulb soak is to be used the Puratized Agricultural Spray followed by demeton seems best. Puratized Agricultural Spray apparently does not affect the action of demeton.

Literature Cited

1. GILL, D. L., FLOYD F. SMITH, and PHILIP BRIERLEY. 1955. Necrotic fleck of Easter lilies. Flor. Rev. 117 (3026): 31-32.
2. LEBEAU, F. J. 1946. The eradicant action of a fungicide on Colletotrichum lilii in lily bulbs. Phytopathology 36: 391-393.
3. LEBEAU, F. J. 1947. A fungicide for protecting lily bulbs from infection by Colletotrichum lilii. Phytopathology 37: 194-196.
4. LEBEAU, F. J. 1947. Treatment of lily bulbs for black scale control. Phytopathology 37: 801-808.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE AND GEORGIA AGRICULTURAL EXPERIMENT STATION, TIFTON, GEORGIA

SYMPTOMS OF CACHEXIA IN ORLANDO TANGELO, NONE IN SWEET LIME AND FALSE SYMPTOMS ASSOCIATED WITH PURPLE SCALE INFESTATIONS

T. J. Grant, G. R. Grimm,[1] and Paul Norman[2]

Abstract

Sometimes the causal agents of xyloporosis and cachexia are considered to be the same and at other times different. In the present study buds from seven widely separated sources of bud inoculum produced definite cachexia symptoms in Orlando tangelo and no symptoms of xyloporosis in sweet lime seedling plants. Inner-bark injury associated with presence of heavy purple scale infestations on the bark surface produced cachexia-like symptoms in Orlando tangelo, sweet lime, and Key lime plants.

Xyloporosis, a disease of sweet orange on sweet lime rootstock in Israel, was first described by Reichert and Perlberger (12) in 1934. Similar reactions were observed by Moreira (7) in Brazil in 1938. Cachexia disease of Orlando tangelo in Florida was described by Childs (2) in 1950. Olson (8) reported cachexia in Texas in 1952. Subsequent to 1950 it was demonstrated (3, 5, 9, 10) that a large number of varieties and hybrids of mandarin orange could express symptoms of cachexia. Its presence in citrus in California and Arizona was reported by Calavan et al. (1).

One to 4 or more years are required for production of cachexia and xyloporosis symptoms. The differentiation or similarity of the causal agents of these diseases are based on observations of different plants grown under widely varying field conditions. These variable factors combined with different sources of inoculum, plant materials and methods have made it difficult to determine whether the diseases are caused by different viruses, by virus mixtures, or by strains of one virus.

The results given in the present paper do not settle the issue but do show that bud inoculum from seven field sources in Florida produced positive cachexia symptoms on Orlando tangelo and failed to produce xyloporosis symptoms on sweet lime seedlings. The occurrence of inner-bark symptoms associated with the presence of purple scale on Orlando tangelo, sweet lime, and Key lime are also reported.

MATERIALS AND RESULTS

Buds from seven widely separated sources were used to inoculate 26 Orlando tangelo and 25 Columbia sweet lime seedlings. The buddings were not all made on the same date, but the test ended with the reading made prior to the severe freeze in 1957 when most of the plants were killed. At that time most of the plants had been inoculated for 2 years and 2 months to 2 years and 8 months. The data, however, included four Orlando tangelo and four sweet limes grown in a protected location for 6 years.

Of the 26 Orlando tangelo plants in the tests, 24 showed definite cachexia symptoms, while of the 25 sweet lime plants inoculated from the same bud sources, none showed xyloporosis symptoms.

False Cachexia-like Symptoms Produced by Purple Scale, Lepidosaphes beckii (Newm.)

In the nursery rows there were a total of 16 Orlando tangelo and 16 Columbia sweet lime plants that were healthy, non-inoculated controls. In the course of examination of these trees bark patches were removed. During the readings taken in September 1957 it was noted that some of the sweet lime control plants and inoculated plants had orange-yellow to brown spots in the inner bark that extended to the cambium. The first impression was that xyloporosis and cachexia had been transmitted by budding and that there had been spread in the field to the controls or seed transmission had occurred, as indicated by Childs (4). However, closer examination of the seedling trees revealed that the abnormal inner-bark discoloration was closely associated with purple scale on the outer bark surface. The inner-bark symptoms were often

[1] Plant Pathologists, Crops Research Division.
[2] Entomologist, Entomology Research Division, Agricultural Research Service, United States Department of Agriculture, Orlando, Florida.

confined to points immediately below a visible mass of scale and did not extend beyond the obviously infested area. Likewise the purple scale was associated with similar inner-bark symptoms on non-inoculated Key lime seedlings growing in the same field planting.

It was evident that inner-bark injury associated with purple scale infestation might easily be confused with the early stages of cachexia infection on Orlando tangelo or might be interpreted as xyloporosis infection on sweet lime. The inner-bark symptoms associated with scale would not be readily confused with the more advanced stages of cachexia infection on Orlando tangelo. The latter has distinct, dark-gum deposits in the inner bark (3). It was also noted that the scale or associated discoloration did not occur below the ground line, whereas cachexia symptoms on susceptible rootstocks are not so limited. Controlled infestations with purple scale on Columbia sweet lime and Orlando tangelo seedlings produced (false) cachexia-like symptoms on these hosts in 13 months. Further tests of the effect of purple scale on inner-bark symptoms on citrus plants are to be reported by Fruit Insects Section of the Entomology Research Division.

DuCharme and Knorr (6) observed pitting in Rough lemon root sprouts in Argentina but the pits were correlated with the presence of the adults of California red scale, Aonidiella aurantii (Mask).

DISCUSSION

The similarities and variations of inner-bark symptoms of cachexia and xyloporosis have been noted (3, 4). The transmissibility of cachexia and its virus nature have been established (3). The symptoms of xyloporosis have been described in detail (12). Controlled tests of transmission of xyloporosis in Israel have been started, but final results have not been reported (11).

Since 1950 there has been appreciable increase in the number of investigations of cachexia and xyloporosis, and this work has been carried out in several widely separated citrus areas of the world. The comparison of results from these investigations has been based largely on described similarities or differences in symptoms and host reactions.

In the present test the Orlando tangelo seeds were from two trees, and the Columbia sweet lime from a single tree at the U. S. Experimental farm planting. The Columbia sweet lime seed source was the same as that used in experimental work in Brazil and there showed symptoms judged to be xyloporosis in one of the inoculation tests carried out. The results of the current tests where seven different field sources of inoculum produced distinct cachexia symptoms on Orlando tangelo but failed to produce xyloporosis symptoms on Columbia sweet lime suggest that these diseases may be caused by different viruses or at least by distinct strains or strain mixtures of a virus.

It seems likely that some confusion and doubt as to the identity of xyloporosis and cachexia will continue until inoculum determined to be free of other viruses is obtained and comparisons are made by uniform methods on test plants under comparable conditions.

Evidence presented here indicates that purple scale causes inner-bark injuries on Orlando tangelo and sweet lime that can be confused with initial cachexia symptoms. Recognition of such symptoms in the presence of purple scale may prevent misinterpretation.

Literature Cited

1. CALAVAN, E. C., J. B. CARPENTER, and L. G. WEATHERS. 1958. Observations on distribution of cachexia of citrus in California and Arizona. Plant Disease Reptr. 42: 1054-1056.
2. CHILDS, J. F. L. 1950. The cachexia disease of Orlando tangelo. Plant Disease Reptr. 34: 295-298.
3. CHILDS, J. F. L. 1951. Cachexia, a bud transmitted disease, and the manifestation of phloem symptoms in certain varieties of citrus, citrus relatives and hybrids. Florida State Hort. Soc. 64: 47-51.
4. CHILDS, J. F. L. 1956. Transmission experiments and xyloporosis-cachexia relations in Florida. Plant Disease Reptr. 40: 143-145.
5. CHILDS, J. F. L., G. R. GRIMM, T. J. GRANT, L. C. KNORR, and G. NORMAN. 1955. The incidence of xyloporosis (cachexia) in certain Florida citrus varieties. Proc. Florida State Hort. Soc. 68: 77-82.
6. DuCHARME, E. P., and L. C. KNORR. 1954. Vascular pits and pegs associated with diseases in citrus. Plant Disease Reptr. 38: 127-142.

7. MOREIRA, S. 1938. Xyloporosis. Hadar 11: 234-237.
8. OLSON, E. O. 1952. Investigations of citrus rootstock diseases in Texas. Proc. Rio Grande Valley Hort. Inst. 6: 28-34.
9. OLSON, E. O. 1954. Some bark and bud union disorders of mandarin and mandarin-hybrid rootstocks in Texas citrus plantings. Proc. Am. Soc. Hort. Sci. 63: 131-136.
10. OLSON, E. O., and A. V. SHULL. 1956. Exocortis and xyloporosis bud-transmission virus diseases of Rangpur and other mandarin-lime rootstocks. Plant Disease Reptr. 40: 939-946.
11. REICHERT, I., A. BENTAL, and I. YOFFE. 1956. Transmission experiments on the tristeza and xyloporosis diseases of citrus. Ktavim 6: 69-75.
12. REICHERT, I., and J. PERLBERGER. 1934. Xyloporosis, the new citrus disease. Hadar 7: 163-167, 172, 183-202.

CROPS RESEARCH DIVISION AND ENTOMOLOGY RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, ORLANDO, FLORIDA

REACTION OF CABBAGE VARIETIES AND CLUBROOT-RESISTANT LINES TO ROOT-KNOT NEMATODES

N. N. Winstead

Abstract

Plants of the cabbage varieties Badger Market, Copenhagen Market, Early Round Dutch, Jersey Queen, and Oakview were susceptible to Meloidogyne incognita incognita, M. incognita acrita, M. arenaria arenaria, M. javanica, and M. hapla. Clubroot-resistant lines were also susceptible. No differences in the susceptibility of the various lines and varieties were noted. Root-knot indices of plants inoculated with M. arenaria arenaria were lower than those of plants inoculated with M. incognita incognita, M. incognita acrita, and M. javanica. All varieties and lines were affected less severely by M. hapla than by the other species and subspecies tested.

In eastern North Carolina root-knot nematodes (Meloidogyne spp., Goeldi, 1887) are of little importance in the spring cabbage crop. However, they may cause much damage in the seedling and young plant stages of the fall crop which is seeded directly in the field during late summer. Under these conditions nematodes are particularly active. While these nematodes can be largely controlled with nematocides, varieties resistant to the disease would be valuable for late summer and fall plantings in North Carolina.

Since both clubroot and root knot cause hypertrophy and hyperplasia in roots of cabbage plants, the major objective of this study was to determine whether clubroot-resistant lines were resistant or susceptible to five Meloidogyne spp. (M. incognita incognita (Kofoid and White, 1919) Chitwood, 1949; M. incognita acrita Chitwood, 1949; M. hapla Chitwood, 1949; M. arenaria arenaria (Neal, 1889) Chitwood, 1949; and M. javanica (Treub, 1885) Chitwood, 1949). In addition, the reaction of certain varieties grown widely in North Carolina to each of these five species and subspecies was determined. The Golden Acre variety previously was reported susceptible to the five nematode populations used in this study[1].

MATERIALS AND METHODS

Inoculum used was increased from single egg-mass cultures on Rutgers or Homestead 24 tomato plants, using techniques previously described[1]. Infested soil containing tomato roots bearing egg masses was used as inoculum. Cabbage seedlings were grown in steam disinfested soil and transplanted to infested soil. Root-knot ratings were made 3 months after inoculation by washing the soil from the roots and examining the roots macroscopically. The following rating system was used : 0 -- no mature females with egg masses; 1 -- light infection with only an occasional female egg mass; 2 -- light infection with mature females and a few egg masses seen easily with the naked eye; 3 -- moderate infection with mature females and egg masses moderately abundant; 4 -- severe infection with mature females and egg masses abundant; and 5 -- very severe. Plants rated as 0, 1, or 2 are considered resistant.

EXPERIMENTAL RESULTS

Varieties and breeding lines of cabbage inoculated with root-knot nematodes included Oakview, Early Round Dutch, Copenhagen Market, Badger Market, Jersey Queen, Wisconsin clubroot-resistant lines 192255L and 1922395413; three North Carolina clubroot-resistant lines (selected from 192255L) N. C. 3, 1958-6, and 1958-7; and the F_2 population of a cross between Oakview and 192255L. A minimum of 30 plants of each variety or line was inoculated with M. incognita incognita, M. incognita acrita, M. arenaria arenaria, M. javanica, and M. hapla. Each of the varieties and lines tested were susceptible to each species tested. Root-knot indices are given in Table 1. Each of the varieties was less severely affected by populations of M. arenaria arenaria than by the populations of M. incognita incognita, M. incognita acrita, and M. javan-

[1] Sasser, J. N. 1954. Identification and host parasite relationships by certain root-knot nematodes (Meloidogyne spp.). University of Maryland Agr. Exp. Sta. Tech. Bull. A-77. 51 pp.

Table 1. Average root-knot indices of cabbage lines and varieties inoculated with five Meloidogyne species and subspecies.

Variety or line	Average root-knot index[a]				
	M. incognita incognita	M. incognita acrita	M. arenaria arenaria	M. javanica	M. hapla
Badger Market[b]	4.6	4.8	3.7	4.5	3.3
Copenhagen Market[b]	4.8	4.6	3.6	4.7	3.4
Early Round Dutch[b]	4.6	4.7	4.2	4.7	3.4
Jersey Queen[b]	4.8	4.5	4.1	4.6	3.3
Oakview[b]	4.6	4.4	4.1	4.7	3.5
Wisc. 192255L[c]	4.8	4.8	3.6	4.5	3.3
Wisc. 1922395413[c]	4.6	4.6	3.9	4.8	3.5
N. C. 3[d]	4.8	4.6	4.1	4.6	3.3
1958-6[d]	4.7	4.3	3.7	4.5	3.3
1958-7[d]	4.8	4.6	4.0	4.5	3.4
F_2 (Oakview x 192255L)	4.7	4.5	4.3	4.7	3.3

[a] Rated on a 0, 1 , 2, 3, 4, 5 scale; 0 = no infection, 5 = very severe infection.
[b] Furnished by Associated Seed Growers, Inc.
[c] Furnished by J. C. Walker, University of Wisconsin.
[d] Selections from 192255L.

ica used in these studies. In addition, all varieties and lines appeared to be more tolerant to M. hapla than to the other species and subspecies. No differences in the susceptibility of the varieties and lines was observed.

DISCUSSION

Both the clubroot and root-knot diseases are characterized by galls or enlargement of the roots. However, clubroot-resistant lines were as susceptible to root-knot nematodes as were clubroot-susceptible varieties. Root enlargements incited by nematodes on clubroot-resistant lines were as numerous and as large as those on clubroot-susceptible varieties; hence, resistance to these two diseases does not appear to be correlated. Therefore the possibility is suggested either that 1) factors produced by the two incitants which stimulate hyperplasia and hypertrophy also differ, or 2) if these substances are identical, the factors responsible for resistance to clubroot in cabbage are unrelated to gall formation.

The reactions of the cabbage varieties and lines to the five Meloidogyne spp. and subspecies used in these tests were essentially similar to those previously reported by Sasser[1] for the Golden Acre variety.

DEPARTMENT OF PLANT PATHOLOGY, NORTH CAROLINA STATE COLLEGE, RALEIGH, NORTH CAROLINA

A STUDY OF ERYSIPHE GRAMINIS RACES ON BARLEY IN WEST VIRGINIA[1,2]

S. C. Shih, Edward S. Elliott and Collins Veatch

Abstract

A new race of Erysiphe graminis f. sp. hordei and seven other races reported previously were isolated from barley grown at Morgantown, West Virginia in 1958. Races were identified on the basis of their pathogenicity on the six standard differential varieties Black Hull-less, Chevron, Goldfoil, Heil's Hanna 3, Nepal, and Peruvian. It is proposed that the new race be designated race 22. Races 8 and 9 are prevalent at Morgantown, as determined by 55 isolations.

Powdery mildew of barley, caused by Erysiphe graminis DC. f. sp. hordei Machal, is considered one of the more serious diseases of this crop in the winter barley-producing area of the United States. Most of the varieties currently grown in West Virginia and adjacent States are not highly resistant to this disease. This has been attributed primarily to the spread of new, or previously rare, races of the pathogen. Reports of studies on the races of the fungus have recently been reviewed by Moseman (1). The present investigation was conducted to identify the races of powdery mildew obtained from barley grown at Morgantown, West Virginia. This information is of interest to barley breeders in connection with the development of varieties resistant to powdery mildew.

The races used in this study were collected at the Agronomy Farm of West Virginia University at Morgantown. Single races were isolated either from conidia on living diseased plants or from cleistothecia on mature or dried leaves. Single pustule isolates were used for inoculation of standard differential varieties. Seedlings of the six standard differentials for races of barley powdery mildew (Black Hull-less (C.I. 666), Chevron (C.I. 1111), Goldfoil (C.I. 928), Heil's Hanna 3 (C.I. 682), Nepal (C.I. 959), and Peruvian (C.I. 935)) were inoculated in the first - or second-leaf stage by shaking conidia onto them from heavily infected plants. During inoculation and incubation the seedlings were grown in isolation cages in a room equipped with day-light fluorescent tubes; the seedlings received about 100 foot candles of light. The temperature was maintained constant at approximately 20°C. The reactions of the seedlings to the various races were recorded when the development of the disease symptoms had reached its maximum, usually 10 days after inoculation. Seedling reactions were recorded in five categories of severity in accordance with the scale described by Moseman (1). The five categories are 0, 1, 2, 3, and 4 which indicated immune, highly resistant, moderately resistant, moderately susceptible, and highly susceptible, respectively. Race identification was made in every trial only when the reaction was distinct on each differential variety. The pure races were maintained by the transfer of conidia to susceptible seedlings of the variety Heil's Hanna 3 at intervals of 20 to 25 days. Plants were grown in 3-inch clay pots under lamp chimneys plugged with cotton.

The results of this study revealed the presence of seven previously reported physiological races of E. graminis f. sp. hordei. In addition, one other race was isolated which, so far as is known, has not been reported previously. The pathogenicity of 22 isolates from conidia and 33 isolates from cleistothecia was determined. The frequency with which each race occurred among these 55 isolates is shown in Table 1.

[1] Published with the approval of the Director of the West Virginia Agricultural Experiment Station.
[2] Based in part on the M. S. thesis of the senior author.

Table 1. Races of Erysiphe graminis f. sp. hordei identified in 55 isolates from barley grown at Morgantown, West Virginia.

Source of race	Races								Total isolates
	8	9	11	14	16	19	20	Unknown	
Conidia	9	6	3	2	-	-	-	2	22
Cleistothecia	13	6	3	4	2	4	1	-	33
Total	22	12	6	6	2	4	1	2	55

It is evident from Table 1 that some of the races occurred much more frequently than others. Race 8 was obtained 22 times, and race 9 was obtained 12 times in 55 isolations.

The unknown race of the fungus found in the present study was isolated from conidia on an infected plant of the variety Goldfoil. It is proposed that this previously unreported race be designated race 22. The reactions of the six differential varieties to this race are as follows: Black Hull-less, 3; Chevron, 4; Goldfoil, 4; Heil's Hanna 3, 4; Nepal, 1-2; and Peruvian, 3.

Except for its increased pathogenicity on the variety Goldfoil, race 22 gave the same types of severity reaction on the differential varieties as race 20, described by Powers and Moseman (3). Race 21 described by Moseman (2) differs from race 22 primarily in its greater pathogenicity on the differential variety Nepal.

Literature Cited

1. MOSEMAN, J. G. 1956. Physiological races of Erysiphe graminis f. sp. hordei in North America. Phytopathology 46: 318-322.
2. MOSEMAN, J. G. 1959. Strain dynamics of powdery mildew of barley, Erysiphe graminis f. sp. hordei, in North America. Plant Disease Reptr. 43: 1004-1009.
3. POWERS, H. R., Jr., and J. G. MOSEMAN. 1957. Pathogenic variability within cleistothecia of Erysiphe graminis. Phytopathology 47: 136-138.

WEST VIRGINIA AGRICULTURAL EXPERIMENT STATION, MORGANTOWN

NITROGEN FERTILIZATION ON THE RESPONSE OF SOYBEANS INFECTED WITH HETERODERA GLYCINES[1]

J. P. Ross[2]

Summary

Split applications of 40, 120, and 210 pounds of nitrogen (NH_4NO_3) per acre to plots heavily infested with Heterodera glycines caused striking increases in soybean yield and final nematode populations. Yield increases from plots fertilized with 120 and 210 pounds of nitrogen per acre were significant. The nematode population in plots receiving the highest rate of nitrogen was significantly greater than that in non-fertilized plots. This response is attributed to stimulation of root growth which provided more multiplication sites for the nematode. Nitrogen fertilization decreased soybean root nodulation.

Infection of soybean (Glycine max) and pea (Pisum sativum) roots by nematodes of the genus Heterodera reduces nodulation by Rhizobium japonicum. Oostenbrink (5) and Jones and Moriarty (2), working with the pea root nematode, Heterodera göttingiana Liebscher, postulated that the application of nitrogen fertilizer to infested soil might counteract the reduction of nodule formation on peas. Oostenbrink also stated that this practice may prove valuable in growing peas in infested land.

Ichinohe and Asai (1) recognized a close inverse relationship between severity of infection by Heterodera glycines Ichinohe and root nodulation on soybeans. They mentioned that soybeans grown on heavily infested land do not exhibit the typical chlorotic symptom of the cyst nematode disease when fertilized with large amounts of nitrogen. The chlorotic symptom on soybeans infected with H. glycines has been observed in North Carolina since the discovery of the cyst nematode in 1954. However, in the Mississippi Delta apparently this chromatic symptom has developed less frequently and has been less pronounced on soybeans heavily infected by the cyst nematode.

Although these reports indicate that nitrogen fertilization may alleviate the symptoms and damage of the cyst nematode disease on peas and soybeans, experimental data are lacking. This report summarizes information obtained during the summer of 1958 on 1) the response of soybeans growing in heavily infested soil to supplemental inorganic nitrogen, and 2) cyst nematode populations under these conditions.

METHODS AND MATERIALS

Individual plots were 18 x 15 feet, contained five rows, and were surrounded by a 3-foot border. Treatments were randomized within blocks and replicated 10 times. Ammonium nitrate was broadcast by hand 9 days before planting at the following rates of nitrogen: 1) 0, 2) 20, 3) 60, and 4) 150 pounds per acre. Approximately 6 weeks after planting of the soybeans (var. Jackson), additional nitrogen was applied as a sidedressing to treatments 2, 3, and 4 at 20, 60, and 60 pounds per acre, respectively.

Five weeks after planting, plant height data and nodulation indices were recorded for eight randomly selected plants from each plot. Yield data were obtained from soybeans harvested on November 11 from the middle three rows of each plot.

Nematode assays of the soil were made on October 1. Composite soil samples were taken to a depth of 6 to 8 inches from the middle three rows of each plot with a 1-inch soil tube. One pint of thoroughly mixed soil from each plot was washed through a 20-mesh screen, and the resulting suspension was thoroughly stirred and immediately decanted through a 60-mesh screen which retained the cysts. The remaining sediment was suspended again, stirred, and decanted through the 60-mesh screen. The material on the screen was forcibly sprayed, transferred to a Baermann funnel, and incubated 10 days before the larvae were counted.

---

[1] The author is indebted to the personnel of the North Carolina Horticultural Crops Research Station at Castle Hayne, under the supervision of J. M. Jenkins, Jr., for their assistance in the maintenance of the experimental plots described herein.

[2] Plant Pathologist, United States Department of Agriculture, Agricultural Research Service, Crops Research Division; and North Carolina Agricultural Experiment Station.

RESULTS

Growth 5 weeks after planting indicated that plant response to the initial nitrogen application was manifested early in plant development (Table 1). Nodulation was significantly reduced in all plots receiving nitrogen and decreased with increasing nitrogen applications. Inhibition of legume nodulation by combined nitrogen was reported earlier (4). Plants in control plots were weakly nodulated.

Table 1. Soybean cyst nematode larvae counts, plants heights, nodulation indices, and yields from soybean plants grown in the presence of various amounts of inorganic nitrogen (NH_4NO_3) in field plots.

Nitrogen added per acre[a] (in pounds)			Larvae per pint of soil[b] (number)	Plant height[c] (inches)	Nodulation index[c]	Yield (grams)
First application	Second application	Total				
0	0	0	405	4.6	2.6[d]	488
20	20	40	591	4.9	2.0*	740
60	60	120	704	6.0**	1.8**	1007**
150	60	210	1217**	6.6**	1.1**	1322**
LSD 1 percent			430	0.4	0.7	361
5 percent			318	0.3	0.5	267

* and ** Differences significant at 5 percent and 1 percent level, respectively.

[a]First application 9 days before planting; second application 6 weeks after planting.

[b]Data taken on October 1.

[c]Data taken 5 weeks after planting.

[d]1, no nodules per plant; 2, 1-3 nodules per plant; 3, 4-6 nodules per plant.

Plant growth throughout the season was directly related to the amount of nitrogen applied. In late summer most of the plants in plots receiving the high rate of nitrogen displayed some chlorosis. However, the intensity and time of appearance of chlorosis were related to the amount of nitrogen applied, that is, the more nitrogen applied the later and less severe the yellowing. In certain plots receiving no nitrogen, growth was extremely poor and most of the plants died.

Nematode populations in the soil in the fall were correlated with the rate of nitrogen application (Table 1). Although the average nematode population in plots receiving 210 pounds of nitrogen per acre is significantly greater than that of the control, the populations in plots receiving lower rates of nitrogen were not; however, the trend is evident.

Soybean yields were positively correlated with the rate of the nitrogen application (Table 1). While the yield from plots receiving 40 pounds of nitrogen per acre is not statistically different from that of the control, it does, nevertheless, approach significance.

DISCUSSION

The data herein reported substantiate the postulations and observations of previous workers (1, 2, 5) and support the idea that the chlorosis of soybeans infected with H. glycines results from a nitrogen deficiency caused by the preclusion of nodulation by R. japonicum.

The possibility that soybeans would also respond to nitrogen when grown in noninfested soils should be considered. Soybeans are not usually fertilized with nitrogen in North Carolina and little or no yield response was noted to applications of 100 pounds per acre of nitrogen (NH_4NO_3) during 3 years of soybean fertilizer trials at several locations (3). In light of these results it seems tenable that in the present experiment the applied fertilizer was supplying the nitrogen normally fixed by root nodule bacteria. Thus, in an experiment of this nature the results may also be affected by factors other than merely the cyst nematode population level. Conditions limiting the availability of nitrogen in the soil, such as the amount of undecomposed organic matter, aeration, activity of microorganisms, and so forth, may assume importance in the disease syndrome of soybeans infected with H. glycines.

The positive correlation between soybean yield and final nematode population ($r = +.550$) in this experiment is probably a reflection of the more appropriate correlation between reproduction of the nematode and root growth of the nitrogen-fertilized soybeans. Since heavy infection by the cyst nematode results in virtually no nodulation of the soybean plant, the role played by the supplemental nitrogen in increasing the nematode population appears to be one of stimulating root growth that provided more sites for multiplication of the nematode.

The sparse nodulation on nonfertilized soybeans is probably the result of cyst nematode activity, while that on soybeans receiving increasing amounts of nitrogen probably was primarily related to the nitrogen inhibition of nodulation. The nematode may also have contributed to some of the nodule inhibition in the fertilized plots.

It does not appear feasible that the application of nitrogen will serve as a practical control measure for this nematode disease in the commercial production of soybeans. However, in special cases it may be of value in increasing yields. Other legumes such as peas and clover infected with Heterodera sp. may react in a manner similar to soybeans in their response to nitrogen applications.

From these results it would seem that the chlorotic symptoms on H. glycines-infected soybeans are dependent on a rather low availability of nitrogen in the soil, and that these symptoms may not develop in soybeans grown in infested soils naturally rich in available nitrogen.

Literature Cited

1. ICHINOHE, M., and K. ASAI. 1956. Studies on the resistance of soybean plants to the nematode, Heterodera glycines. I. Varieties, "Daiichi-Hienuke" and "Nangun-Takedate". Research Bull. Hokkaido Natl. Agr. Exp. Sta. 71: 67-79.
2. JONES, F. G. W., and F. MORIARTY. 1956. Further observations on the effects of peas, beans and vetch upon soil population levels of pea root eelworm, Heterodera göttingiana Liebscher. Nematologica 1: 268-273.
3. NELSON, W. L., and C. D. WELCH. 1947. Nutritional studies with soybeans. North Carolina State Exp. Sta. Annual Rept. Unpublished.
4. NUTMAN, P. S. 1956. The influence of the legume in root-nodule symbiosis. A comparative study of host determinants and functions. Biol. Revs. 31: 109-151.
5. OOSTENBRINK, M. 1955. Een inoculatieproef met het erwten cystenaaltje, Heterodera göttingiana Liebscher. Tijdschrift over Plantenziekten 61: 65-68.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, AND NORTH CAROLINA AGRICULTURAL EXPERIMENT STATION, RALEIGH

SURVIVAL OF LOOSE SMUT IN BARLEY SEED[1]

Karl D. Fezer[2]

Ustilago nuda (Jens.) Rostr. has been reported to survive up to 11 years in barley seed[3]. However, there have been no reports on whether the percentage of smut-infected seed changes with time because of loss of viability of the smut or because of differential survival between infected and non-infected seed. Twenty-nine varieties or strains of barley differing in susceptibility to loose smut were inoculated in 1955 by the air-blast method[4]. The seed from 8 to 10 inoculated heads of each variety was planted in 6-foot head-rows in 1956, and 4 to 5 head-rows of each variety were planted in 1959. The average percent of heads infected in individual varieties ranged from 0 to 70. The average for all 29 varieties was 33 percent in 1956 and 34 percent in 1959. Therefore, there is no evidence for a difference in survival between barley seed and the smut inside it, or between infected and non-infected seed, during the first 4 years following infection.

DEPARTMENT OF PLANT PATHOLOGY AND BOTANY, INSTITUTE OF AGRICULTURE, UNIVERSITY OF MINNESOTA, ST. PAUL, MINNESOTA

---

[1]Paper No. 4267, Scientific Journal Series, Minnesota Agricultural Experiment Station.
[2]Instructor, Department of Plant Pathology and Botany, University of Minnesota, St. Paul, Minnesota.
[3]Porter, R. H. 1955. Longevity of Ustilago nuda in barley seed. Phytopathology 45: 637-638.
[4]Moore, M. B., and D. E. Munnecke. 1954. An air blast method for rapid inoculation of wheat and barley with loose smut. (Abst.) Phytopathology 44: 499.

CROWN RUST ON BARLEY IN MINNESOTA[1]

R. W. Lutey and R. P. Covey[2]

In May 1959 urediospores of Puccinia coronata (Cda.) f. sp. secalis Peturson were collected from Traill barley in the field at St. Paul, Minnesota. This constitutes the first report of this disease on barley grown in Minnesota.

The rust was most prevalent on barley growing in plots approximately 10 feet from buckthorn (Rhamnus cathartica), the alternate host of the organism. A large number of aecial clusters were observed on the buckthorn in the spring, prior to the infection of the barley.

The urediospores collected were used to inoculate, in the greenhouse, Kindred, Montcalm, Traill, and Oderbrucker barleys, Gopher and Victory oats, quackgrass (Agropyron repens), Prolific spring rye, and a mixture of winter ryes containing Dominant, Sandgaste, Heines Hellkorn, and Caribou varieties. The greenhouse inoculations were repeated three successive times, using the urediospores produced from the previous inoculations as inoculum each time.

Results were consistent through each inoculation. All barley varieties were moderately susceptible. The spring rye, and a mixture of winter ryes were very susceptible. Both Gopher and Victory oats were immune. Quackgrass was moderately susceptible. The infection type produced on barley in the greenhouse was the same as that observed on barley infected in the field. Infection of volunteer barley in the same location as the earlier field infection was observed in late September, 1959.

Urediospores and teliospores obtained from collections in the field corresponded to the description published by Peturson[3], as did the disease reactions obtained from inoculations made in the greenhouse.

DEPARTMENT OF PLANT PATHOLOGY AND BOTANY, INSTITUTE OF AGRICULTURE, UNIVERSITY OF MINNESOTA, ST. PAUL, MINNESOTA

---

[1]Paper No. 4268, Scientific Journal Series, Minnesota Agricultural Experiment Station, St. Paul, Minnesota.
[2]Research fellow and research assistant, respectively, Department of Plant Pathology and Botany, University of Minnesota, St. Paul, Minnesota.
[3]Peturson, B. 1954. The relative prevalence of specialized forms of Puccinia coronata that occur on Rhamnus cathartica in Canada. Can. J. Botany 32: 40-47.

BRIEF NOTE

OAK WILT IN OKLAHOMA AND ARKANSAS

By A. F. Verrall,
E. R. Toole,
and P. C. Lightle

Oak wilt, caused by Ceratocystis fagacearum (Henry) Hunt, was found for the first time in Oklahoma in June 1959. Two wilting oaks were observed during a limited aerial and ground survey of eastern Oklahoma. The causal fungus was isolated by Dr. C. L. Wilson of the University of Arkansas from samples taken from one of the trees. Both trees were in Adair County, only a few miles west of the oak wilt area in Arkansas. Indications are that the incidence of wilt in eastern Oklahoma is low.

Aerial surveys in northern Arkansas in 1958 and 1959 disclosed oak wilt only in the general area in which it was originally found there in 1950 and 1951 (Logan, Crawford, Washington, Madison, Newton, Boone, Baxter, Fulton, Sharp, Independence, Lawrence, and Craighead counties). Since 1951, wilt has been found in only four other counties (Carroll, Randolph, Stone, and Searcy) -- all within the previously known general range. Alarming buildup of oak wilt in Arkansas occurs only in Sharp County in northeast Arkansas and Logan County in west central Arkansas.

UNITED STATES DEPARTMENT OF AGRICULTURE,
FOREST SERVICE, SOUTHERN FOREST EXPERIMENT STATION

CORRECTIONS

REPORTER, October issue (Volume 43, Number 10). Dr. Charles R. Maier has called to our attention an error on his part in the listing of authors for the article, "Cultural and pathogenic variability of Rhizoctonia solani isolates from cotton-growing areas of New Mexico," which appeared on pages 1063-1066. The authors should read "Eugene E. Staffeldt and Charles R. Maier." Dr. Staffeldt is Assistant Plant Pathologist, Department of Biology, New Mexico State University and New Mexico Agricultural Experiment Station.

REPORTER, November issue (Volume 43, Number 11), page 1182. The legend for Figure 3 should read "Ranger alfalfa plants 2 weeks after wound inoculation, etc." rather than "Ranger alfalfa plants 2 weeks old after wound inoculation, etc."

REPORTER, November issue (Volume 43, Number 11), page 1198. The name of the second author of the article beginning on this page should be spelled Manuel Carnero, rather than Manuel Carero.